

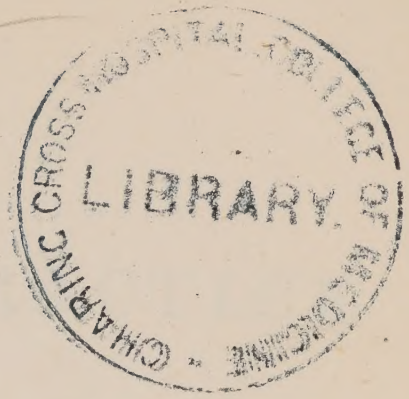


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THE
SCIENCE AND PRACTICE
OF
MEDICINE.

BY
WILLIAM AITKEN, M.D., EDIN.,
PROFESSOR OF PATHOLOGY IN THE ARMY MEDICAL SCHOOL.

IN TWO VOLUMES.
WITH A STEEL PLATE, A MAP, AND ONE HUNDRED AND THIRTY WOOD-CUTS.

VOL. I.

FIFTH EDITION, ENLARGED AND CAREFULLY REVISED,
ADOPTING THE NEW NOMENCLATURE OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

LONDON:
CHARLES GRIFFIN AND COMPANY,
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1868.

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PREFACE TO THE FIFTH EDITION.

A FAMOUS novel-writer of the day has recorded that "a Preface is a pleasant thing to write, whatever it may be to read." To indorse the record, and to write a Preface for the *Fifth* Edition of this Text-book, is very gratifying to the Author.

On the 6th of December, 1866 (twelve months after the publication of the *Fourth* Edition), the Publishers wrote to the Author, saying, "The state of the stock now renders it necessary for us to request you will prepare a *Fifth* Edition."

In accordance with this request, the Author has been engaged during the past fifteen months in a careful revisal of this Work, and the result is that the *Fifth* Edition has increased in bulk upwards of 100 pages.

This increase implies many more considerable changes (which it is hoped are improvements), as well as additions, than can be made obvious in a Preface. They consist, however, mainly, in—

First,—The adoption and incorporation in the Text of the "New Nomenclature of the Royal College of Physicians of London." The plan and basis of this nomenclature and classification of diseases is stated at p. 183, vol. i., and a tabular view of the English portion of the Nomenclature of Diseases is substituted at p. 185, vol. i., for the Nosology of Dr. Farr, which had been used in the previous editions. The reasons for this change are to

be found in the account of the "Present State and Aim of Nosology," at p. 179, vol. i.

Second,—The new *English Nomenclature* of the College has been adopted throughout the text. The *definitions* of diseases and the *foreign equivalents* for their English names have been incorporated where the several diseases are described.

The Author is indebted to the kindness of Dr. Sibson for the privilege of an early copy of the new *Nomenclature* of the College, as it passed through the press, with a view to its being used in this edition.

Third,—The subjects of *Malignant Cholera*, of *Paralysis*, of *Epidemic Cerebro-Spinal Meningitis*, and of *Intestinal Obstruction*, have been entirely re-written.

Fourth,—The subjects of *Progressive Locomotor Ataxy*, *Progressive Muscular Atrophy*, *Glosso-laryngeal Paralysis*, *Aphasia*, and *Dilatation of the Bronchial Tubes*, the application of the *Sphygmograph*, and its tracings in diseases, where it has been of use, are subjects considered for the first time in this Text-book.

As with previous editions, so with this one, the Author has aimed at giving as fully and faithfully as he could the ideas and the views of the more advanced and able writers of the time, ever desirous that his Text-book should be a "representative book" of the Medical Science and Practice of the day, as actually understood and followed by the best men of our Profession.

PREFACE TO THE FOURTH EDITION.

A FOURTH EDITION of this Work having been as suddenly called for to succeed the *Third* as the *Third* Edition was called for to succeed the Second, the Author has again submitted every page to a careful revision. He has endeavoured, as before, by condensing some parts, by re-writing others, and by adding new matter, to render the Work still more worthy of the very favourable reception which it has received from the Profession and from the Medical Press.

That the last two (large) Editions should have been exhausted in less than twelve months, the Author cannot but regard as one of the greatest honours which the Profession could bestow on one of its members; implying, as it may be presumed to do, a certain approbation of the method of the text, and of the management of its matter; and thus also conferring at the same time weight and authority to the Work.

With greater confidence, therefore, the Author trusts he may presume to Dedicate this Edition especially TO STUDENTS OF MEDICINE, in the hope that his Work may deserve and obtain their deference and respect.

ROYAL VICTORIA HOSPITAL,
NETLEY, *September, 1865.*

PREFACE TO THE THIRD EDITION.

THE Second Edition of this Text-book had not been published more than six months when it was exhausted, and the Author found himself suddenly and unexpectedly called upon to prepare a Third Edition. He has accordingly experienced great satis-

faction from so favourable a reception of his Work, on the part of the Profession, and of the Medical Press; and in particular he has been highly gratified by the approbation of the Teachers throughout the Schools of Medicine in England, Scotland, and Ireland, and of the Professors of Medicine in the Universities. From many fellow-workers in the profession he has received most useful hints and obliging communications, to be remembered with gratitude, and from which he hopes this Edition has profited much.

Stimulated by such encouragement, every page of this Edition has been submitted by the Author to a careful revision. He has especially aimed at improving the Work by re-writing some portions, and by condensing others, in order to make room for new material, especially regarding the treatment of diseases. This has not added much to the bulk of the volumes, compared with the previous Edition.

The changes in the nomenclature, and of the doses of remedies as set forth in the *British Pharmacopœia* recently issued by the Medical Council, have been duly attended to, and the remedies have been prescribed in accordance with the requirements of this new Pharmacopœia.

My thanks are especially due to my friend Dr. Thomas Graham Balfour, F.R.S., the Chief of the Statistical Branch of the Army Medical Department, for his kindness in revising and correcting the part on *Medical Geography*. To him, to Dr. Henry Marshall, and to the late Sir Alexander M. Tulloch, science is largely indebted for our knowledge regarding *Medical Geography*; and to the joint reports of these men may fairly be assigned the merit of having first called the attention of the military authorities to the actual condition of the army in regard to health, and to the various deteriorating agencies by which the soldier comes to be affected in different regions of the world. That so distinguished an authority as Dr. Balfour should have been so kind as to revise that portion of my Work which treats of these topics, demands from me the most grateful acknowledgments.

The Author is sorry he cannot meet the views of those friendly critics who desire more positive rules regarding the treatment of diseases than he has considered it judicious to give. The Work has been written with the purest wish to promote the instruction and improvement of the Student of Medicine, and to have stated

dogmatically what is not quite certain could not have a wholesome tendency. The Author believes that the plan and whole scope of this Text-book put it in the power of the Teacher to convey instruction with more ease, and in a shorter space of time, than can be done without a Manual following a *systematic* arrangement, and giving a consistent view of the main facts, doctrines, and practice of Medicine.

ROYAL VICTORIA HOSPITAL,
NETLEY, *September, 1864.*

PREFACE TO THE SECOND EDITION.

IN preparing a Second Edition of the HANDBOOK OF THE SCIENCE AND PRACTICE OF MEDICINE the Author has endeavoured, without material alteration in the general scheme of the Work, to render it more deserving of the confidence of the Student.

The extensive sale of the First Edition, as well as the unsought testimonies to its usefulness which the Author has received from many teachers and practitioners of medicine previously unknown to him, have satisfied the Author that the Work has met the want it was mainly designed to supply—namely, a Text-book for Students, following a systematic arrangement, which would give them a consistent view of the main facts, doctrines, and practice of Medicine, in accordance with the present state of the Science.

That the Work should have been so favourably received demands the most grateful acknowledgments of the Author.

The several sections composing the introductory portion of the former Edition have been somewhat expanded by the addition of important topics relative to Pathology; and, with a view to follow a more natural order in the treatment of the subject, the Introductory sections of the former Edition now form Part I. of this Text-book, and as such are still to be considered as introductory to the succeeding parts.

The whole Work has been carefully revised throughout; numerous additions have been made where topics of importance had been only shortly noticed before; and the paragraphs on the prevention and treatment of diseases have been more fully expressed. The Work, indeed, has been almost entirely re-written; and descriptions of many diseases, omitted in the former edition, are now introduced.

Although still adopting in the main the nomenclature and classification used by the Registrar-General and by the Army Medical Department, yet there are some diseases (such, for example, as *Rheumatism*, *Chronic Bright's Disease*, *Diabetes Mellitus*, and the like) whose pathology now forbids their being considered in the place where they are respectively found in that classification; but otherwise, for the reasons stated in Part II. (and especially between pages 169 to 174), that nomenclature and classification have both been retained as the fundamental part of the system on which the arrangement of the Text-book is based.

Diagrams illustrative of the typical ranges of temperature in febrile diseases are now given for the first time in a Text-book. The Author has been induced to do this from a belief in the great practical importance of the subject, and in the hope that it may lead those who have opportunities for original observation to add to our knowledge of febrile diseases in this direction. These diagrams have been introduced advisedly, after consultation with one who has had great experience as a clinical teacher, and for whose opinion the Author entertains a most profound respect.

Wood-cuts have been introduced wherever it was thought they would render the descriptions in the text more intelligible; and short sections, relative to the main points to be attended to in clinical diagnosis, have been given at the heads of the chapters treating of those "diseases in the course of which lesions tend to be localized."

As with the compilation of the First Edition, so now, the Author has numerous obligations to acknowledge; and, first of all, his best thanks are due to his esteemed friends and colleagues, Professors Longmore, Maclean, and Parkes. To them he is indebted not only for advice on many points, but for information and material which he would not otherwise have had, and all of

which he has endeavoured duly to identify and acknowledge at its proper place in the text.

The Author's thanks are also due to Dr. Sidney Ringer, the Professor of *Materia Medica* in University College, for the use of his Notes on the ranges of temperature in cases of *Rheumatism*, and for permission to publish the details given in the text relative to temperature in that and other diseases.

To his friend Dr. T. M. Anderson, of Glasgow, the Author owes his thanks for the use of wood-cuts illustrative of parasitic diseases of the skin; and to his friend Otto Striedinger, Esq., the Secretary of the Army Medical School, the Author is indebted for many excellent drawings, as well as for other valuable assistance.

Agreeably to facts and doctrines set forth in the second volume of the *Ophthalmic Hospital Reports*, pp. 117 and 120, the Author advised that these volumes should be printed on paper of a yellowish tone; and his thanks are due to the printers for the care, ability, and skill with which the typography of a text containing so many terms purely scientific has been executed.

ROYAL VICTORIA HOSPITAL,
NETLEY, *September, 1863.*

PREFACE TO THE FIRST EDITION.

IN the compilation of this HANDBOOK I have attempted to give a condensed view of the SCIENCE AND PRACTICE OF MEDICINE. It has also been my object to incorporate and connect the more recently established facts which illustrate the *Nature of Diseases* and their *Treatment* with the time-honoured doctrines on which the Science of Medicine has been based.

While the greater portion of the volume is necessarily devoted to a consideration of the *Nature* and *Treatment* of individual diseases, a more comprehensive range of topics has been embraced, under the title of the SCIENCE OF MEDICINE, than it has hitherto been usual to include in text-books.

The introductory sections indicate the more important element of *General Pathology*; and those principles are shortly stated on which the more modern systems of *Nosology* have been founded since the time of Cullen.

The remainder of the volume, arranged in three divisions, treats, in the FIRST PART, of *Systematic Medicine, Nosology, or the Classification of Diseases*, and suggests that the classification of the Registrar-General of England should be adopted. This statistical nosology, originally proposed by Dr. William Farr, has been carefully discussed and revised at the recent meeting of the Statistical Congress held at Vienna, and a *nomenclature* substantially the same is proposed for adoption in all the States of Europe. The fatal cases are to be *registered* on a uniform plan. A definite *classification*, however, is still undetermined; but I am kindly informed by Dr. Farr that a *classification* nearly the same as the English one has been adopted in Bavaria, and is quietly making its way among practical men in Germany. The Austrians, also, as represented by Dr. Hebra, approve of the separation of *Zymotic* diseases from the others.

In PART SECOND, under the head of the *Nature of Diseases, Special Pathology, and Therapeutics*, I have attempted to describe the nature of each disease considered as characteristic of its class. In so doing, each disease or morbid process has been defined, not by a logical definition, but merely by stating prominently its leading characters, so that the student may *at once* distinguish the general features of the disease which he has to study, and which the physician has to treat. Having then established the position of each disease in its *Nosological* and *Pathological* relations, those principles are stated which guide its treatment, and in some instances definite details are given.

In PART THIRD, under the head of *Medical Geography, or the Geographical Distribution of Health and Disease*, a prominent place has been assigned to a most important department of the Science of Medicine—a subject of study hitherto, so far as I am aware, wholly untaught at our medical schools in this country. It was emphatically written by Cabanis and Malte Brun that climate and natural history lost much of their value from the fact that the physical conditions of the surface of the earth had not then been described in relation to these studies—a deficiency now in a great measure supplied by the labours of Humboldt,

Berghaus, and Johnston. So also it may be stated that the NATURE OF DISEASES and their distribution on the globe require that they should be studied in relation to the physical condition of the earth's surface, and to the variation of their *types* in the different regions of the earth. The geographical distribution of Health and Disease in relation to Physical Geography is a branch of the Science of Medicine rapidly and justly growing in importance; and in one department—that of *Sanitary Science*—is beginning to yield most important fruits. To Dr. Mühry in Germany, M. Boudin in France, and Mr. Keith Johnston in this country, the Science of Medicine is largely indebted for the elucidation of this important topic; of which I have attempted to give a sketch, illustrated by Mr. Johnston's map, indicating some of the more useful directions which the study may take.

In my attempt to accomplish this design I have many obligations to acknowledge. In the first instance, the work has its origin in an Article on the "Elementary Principles of Medicine," contributed to the *Encyclopædia Metropolitana* by the late Dr. Robert Williams, a distinguished physician of St. Thomas's Hospital, London. This Article contains the elements of his classic work on *Morbid Poisons*, completed about sixteen years ago—a work which "occupies the highest rank in the practical literature of this country;" and his views regarding their nature are here preserved, commencing from page 185 of this Text-book. It was originally intended by my publishers to reproduce that Article, but I deemed it necessary entirely to re-write and re-model the whole, retaining the statements of facts, and such illustrations as appeared to be of sufficient importance. While I have collected information from every other available source, the limits of this volume prevent me doing more than simply stating at the end of each paragraph the name of the Author from whose writings the statements have been compiled. If I have correctly interpreted and stated the doctrines taught by the veteran labourers and original investigators in the fields of medical experience and research, the names of those of whose writings I have freely and largely availed myself will furnish a sufficient guarantee that the matter I have attempted to communicate is at least orthodox. Much valuable material I have also to acknowledge from anonymous contributors to the pages of the *Medical Journals*. For access to books and libraries I beg

especially to express my thanks to Dr. Sieveking, Mr. Martin, Sir James Clark, Dr. Steele, and to the Library Committee of the Royal College of Surgeons. Lastly, my best thanks are due to Dr. Steele, Superintendent of Guy's Hospital. Notwithstanding the unceasing demands upon his time which the onerous duties of his office entail upon him, he has kindly revised the sheets as they passed through the press;—for their numerous imperfections I alone must bear the responsibility.

LONDON, *12th October, 1857.*

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ADDITIONS AND CORRECTIONS.

Chap. II.—In the new Nomenclature, commencing p. 185, the following names have been added since the sheet was printed—namely: *Beri-beri*, *Delhi boil*, *Aleppo evil*, *Syphilitic disease of testicle*, and *Paralysis from Lathyrus (sativus)*.

Where the term *Hydatid* occurs, or where the name of a parasite is given amongst the local diseases, the general name of “*Parasitic disease*” has been substituted, returning the case according to the list of parasites which are described in Chapter XII. of this vol., p. 862.

ERRATA.

Page 37, top line, and again at p. 50, line 7, for “one-tenth R. = 1° Fahr.,” read “equal to 225 Fahr.”

Page 120, for “*Amyloid*,” substitute the term “*Lardaceous*,” as its equivalent.

THE SCIENCE AND PRACTICE OF MEDICINE.

PART I.

TOPICS RELATIVE TO PATHOLOGY.

CHAPTER I.

OF MEDICINE AS A SCIENCE AND AS AN ART; ITS OBJECTS AND ITS EXTENT.

THE study of MEDICINE is prosecuted under two relations, namely, as a *Science* and as an *Art*. MEDICINE, considered as a *Science*, takes cognizance of all that relates to our knowledge of diseases; and, especially, of the circumstances under which they become developed, of the conditions of their existence, of their nature and of their causes in the widest sense of these terms. Considered as an *Art* (in so far as Medicine has that practical value), its object is to distinguish, to prevent, and to cure diseases.

The object and aim of MEDICINE as an *Art* is to alleviate human suffering, and to lengthen out human existence, by warding off or by modifying disease "as the greatest of mortal evils," and by restoring health, and even at times reason itself, "as the greatest of mortal blessings." In other words, the practical view required to be taken of MEDICINE is, that "it is the art of understanding the nature of diseases, so as to appreciate their causes, and to prevent their occurrence when possible; to promote their cure, or to relieve them when they occur" (BIGLOW).

Many branches of human knowledge are combined in the constitution and elucidation of the *Science*; and the practice of

Medicine as an *Art* ought to be founded on principles and facts of universal, or at least of extensive applicability.

A consideration of the different topics which together make up the Science of Medicine suggests a division of the subject into the following departments, namely:—(1.) PHYSIOLOGY, which embraces the study of the healthy functions of which the human body is the seat or instrument; (2.) PATHOLOGY, subdivided into *Special Pathology* and *General Pathology*, which together embrace a consideration of everything relative to the existence and nature of diseases; (3.) THERAPEUTICS, which expounds the various actions of remedies upon the diseased economy, or the means by which Nature may be aided in her return to health; (4.) HYGIÈNE, which embraces a consideration of the means of preventing disease, or, in other words, of preserving health.

Physiology, General Pathology, Therapeutics, and Hygiène are sometimes designated indifferently by the titles of the "*Institutes*," the "*Institutions*," or the "*Theory of Medicine*."

These departments of science are all preliminary subjects of study, and constitute a necessary and appropriate introduction to the Practice of Physic, in which Special Pathology and the treatment of special diseases are the leading topics of consideration.

Each of these departments has grown or expanded itself into a great branch of science; and any single section is sufficient of itself to occupy the lifetime of an individual in working out and studying it in detail. It is therefore not possible for the human mind to embrace all of these departments in their whole extent or relations to each other; and, setting aside the consideration of theories and systems, it has been truly observed, "that no man possesses all the pathological knowledge contained in the records of his art" (CHOMEL); and it is, therefore, far less possible to embrace in any single treatise a view of the Science of Medicine in all of these departments.

For the purpose of teaching the Science of Medicine in its application to practice, its elementary principles, as developed in the departments of Pathology, are the most useful guides to the student.

CHAPTER II.

HOW THE PROVINCE OF PATHOLOGY IS MAPPED OUT.

AN inquiry into the nature of diseases embraces a consideration of the following topics:—(1.) *The accurate observation and correct registration of facts in Pathology.* On the efficiency of the machinery devised for these important ends will rest our power to curb the invasion of our science by the guess-work of theory. Such records must be the means eventually of rooting out the traditional errors which so largely pervade medical literature. (2.) *Descriptive Pathology, embracing GENERAL and SPECIAL Pathology.* Special Pathology is intended to comprehend a consideration of the essential nature and origin of particular diseases as they occur in man and animals, and General Pathology to include those more general facts or principles which result from a comparison of particular diseases with each other. Although Special Pathology comes first in the order of Nature, yet, wherever the arrangements for Medical education are complete, General Pathology is taught as an introduction to, or conjointly with, the special study of diseases, just as in other sciences—for example, in chemistry—it is found convenient to give a general view of the principles which have been established by experiment and observation, before entering upon the particular details of the science. All theory in Medicine; all descriptive Pathology; all grounds for rational speculation regarding the nature of diseases, and for the framing of experiments; as well as all maxims of practice which aim at the prevention or cure of diseases, must rest ultimately on observed and recorded facts. *Accuracy of observation* is therefore the first lesson the student has to learn in all methods of investigation, and the lesson is one of paramount importance. The best observations, however, will avail but little unless the observed facts are recorded in such a way as to secure their preservation; and descriptive Pathology mainly concerns itself, in the first instance, with the *Registration of facts*, as embraced,—(1.) In the *History of cases of disease* from their origin to their end. (2.) In the *Statistics of disease*. Such registration includes methods for preserving, in an authentic and

permanent form, the memory of facts in Pathology as they occur. It thus eventually furnishes materials upon which future Pathologists and Statists will build a comprehensive and definite system of scientific Medicine. It will furnish the means of teaching all that is necessarily involved in our notions regarding the nature of diseases. The descriptive Pathology so arrived at considers diseases as they exist, or have existed in man, in the lower animals, or in plants. It considers the conditions under which diseases originate; it considers how far certain conditions are fulfilled before disease establishes itself; and it aims at demonstrating how far such conditions are inconsistent or incompatible with the maintenance of health. Subsequently, with extended information, descriptive Pathology may undertake to assign the conditions which give rise to certain diseases rather than to others. It will eventually define the elements necessary to establish, to originate, or to constitute particular diseases; and will show how the same disease, or class of diseases, may assume various forms, but in all of which definite elements are recognizable. Descriptive Pathology thus aims at determining and describing the essential elements of a disease. (3.) *Speculative Pathology* assumes that we know what a disease is—that we know the effects it produces—that we know the conditions necessary for its existence—that we know its relations to other diseases. It seeks to inquire how certain conditions or circumstances will operate in bringing about disease. It seeks to determine the tendency in the future of a diseased state from certain observed facts in its course, or in the course of similar diseases. Statistical data are thus the main basis of its operations. (4.) *Pathology dictates the maxims of rational practice.* It is in the nature of the science of Pathology that it always ought to be in advance of our certain knowledge regarding the treatment of diseases. It is the basis of rational medicine; for it is rational to know the nature of a disease, in order (1.) To enable us to prevent it; (2.) To understand the principles which ought to guide us in the management of it. Such are the main divisions which the province of Pathology embraces.

It is intended, however, in the first part of this handbook merely to guide the student to notice,—(1.) The *relative* nature of the terms “*Life*,” “*Health*,” “*Disease*,” (2.) How the nature of diseases may be elucidated; (3.) The nature of the morbid

phenomena, symptoms, and signs of disease; (4.) The means and instruments of investigation into the nature and causes of disease; (5.) Some of the more elementary constituents of disease; (6.) Some complex morbid states associated with individual diseases, or with conditions of ill-health (*cachexiæ*); (7.) The modes by which diseases terminate fatally; the types of disease and their tendency to change; (8.) The general treatment of the complex morbid processes.

In the three subsequent parts of this work it is intended to consider some of the details of the science and practice of Medicine, to furnish the student with,—(1.) A nosological system by which to classify and name diseases. (2.) A detailed description of characteristic diseases in the respective classes of that nosological arrangement. In this part a definition (provisional) and a history of the nature of each disease will be given; the probable course and succession of events will be described, and the grounds on which an accurate diagnosis may be made, or a prognosis expected; and, lastly, a detailed account of those rational modes of treatment which are consistent with the established principles of the *Institutes of Medicine*. (3.) An account of what is known relative to the geographical distribution of diseases.

CHAPTER III.

RELATIVE NATURE OF THE TERMS *LIFE*, *HEALTH*, *DISEASE*.

THE word *Disease* is used in a general and also in a specific sense; as when it is said that a person is diseased, without the nature of the affection being stated; or, that he suffers from a particular disease, such as small-pox. Attempts to give a precise definition of the term *Disease* have all been unsuccessful. The relations of the morbid state to the condition of health, and of health to the performance of the vital functions, are of such a kind that they can merely be described in connection with each other, but not defined.

If *life* is understood to imply an active state, resulting from the concurrent exercise of the functions of the body, then there

are conditions of activity and of mutual adaptability of functions and of parts, both as regards body and mind, which are necessary to healthy existence. Our notions of *the conditions of health* have thus considerable latitude. *Health* is merely a name we give to that state or condition in which a person exists fully able, without suffering, to perform all the duties of life. Many degrees of this state are therefore at first sight obvious, from the possession of a feeble existence to the most robust condition of the body; and there are many degrees of feebleness and delicacy of health which we cannot say are due either to disordered or diseased states of the frame. Our notions of *normal life* are thus so extremely indefinite that it is only by a forced abstraction the normal can be separated from the abnormal. Hence also our idea of *disease* is very indefinite; it cannot be separated by any well-defined boundary from our idea of *normal life*, and the two conditions are connected by a kind of debatable border land.

When we regard, therefore, the phenomena of the living state and the conditions of health, we can readily observe when and how *disease* is but A DEVIATION FROM THE STATE OF HEALTH, CONSISTING FOR THE MOST PART IN A CHANGE IN THE PROPERTIES OR STRUCTURE OF ANY TISSUE OR ORGAN, WHICH RENDERS SUCH TISSUE OR ORGAN UNFIT FOR THE PERFORMANCE OF ITS ACTIONS OR FUNCTIONS ACCORDING TO THE LAWS OF THE HEALTHY FRAME.

It is now a received pathological doctrine that *disease* does not consist in any single state or special existence, but is the natural expression of a combination of phenomena, arising out of impaired function or altered structure. All attempts, therefore, to define disease by the use of such terms as "*derangement*," "*modification*," "*alteration*," "*change*" from the pre-existent state of health, show, in the first instance, that very various ideas are attached to the term or to the state; and, secondly, that these terms point to a nosological division into structural and functional disease, rather than to a state common to all forms of disease.

A definition of any state of disease ought to include all the circumstances, whether functional or organic, which constitute the deviation from health; and for very obvious reasons such a definition can only be approximately expressed.

CHAPTER IV.

HOW THE NATURE AND CAUSES OF DISEASES MAY BE
ELUCIDATED.

THE nature of the derangements to which the human body is liable may be studied under the three following aspects:—(1.) As diseases present themselves in individual cases, becoming thereby the subjects of CLINICAL INVESTIGATION and INSTRUCTION—a method of teaching in which the *Natural History* of the disease ought to be a special subject of study; (2.) As they constitute particular genera or species of disease, forming the topics of SPECIAL PATHOLOGY; (3.) As they may be reduced to and studied in their primary elements, forming thereby the science of GENERAL PATHOLOGY.

But, in whatever aspect we may view disease, there is invariably presented to the student the same subjects for investigation, namely,—*First*, The morbid phenomena or symptoms by which we become aware that derangements have taken place in the economy. It is by a mental effort that either the student or the physician converts these symptoms into signs of disease; and hence arises the necessity of studying *Symptomatology* or *Semeiology*. *Second*, The agents by which derangements and diseases are produced, generated, or brought about, constituting the department of *Etiology*. *Third*, the seats or localities of disease, or of derangements, constituting *Pathogeny*. Here the peculiar nature, general forms, and types of disease must be studied, together with varieties in their course, duration, and termination. *Fourth*, The morbid alterations discoverable in the structure of the body before, but more especially after death, constituting *Morbid Anatomy*. These alterations must be studied in connection with the symptoms, the causes, and the course of the disease. *Lastly*, The elementary constituents of diseased products, constituting *Morbid Histology*, must be recognized in the first instance, and contrasted with analogous constituents of the body in the healthy state.

CHAPTER V.

OF MORBID PHENOMENA, SYMPTOMS, AND SIGNS OF DISEASE.

IT has been stated that only by a mental effort is the student or physician able to convert *symptoms* into *signs* of disease. Therefore the idea associated with "*sign*" is of a much more comprehensive kind than that which is connected with the word "*symptom*:" the former implies the possession of more extensive knowledge—a knowledge such that comparisons may be instituted amongst the symptoms which present themselves. Certain symptoms of disease, or of disordered function, are thus recognized to be peculiar, characteristic, or *significant* of a particular morbid state. A symptom is thus converted into a sign, and what is called a *diagnosis* of the disease is made.

Symptoms and signs of disease derive their importance from the fact that they are capable of being connected with lesions of structure or disorders of function; and both of these conditions mutually act and re-act upon each other, and thus they greatly aggravate the expression of general disease. In place of the concurrent exercise of function, and the mutual co-operation of parts in a state of health, both as regards mind and body, we have *symptoms* of disease expressed in various ways, characteristic of the function at fault, and incompatible with the normal existence of the part or organ affected, or of the body generally. From such phenomena the physician makes up his mind,—(1.) As to whether or not disease exists. (2.) How far the condition of the patient is removed from the state of health usual to him. (3.) As to the nature of the disease, and how it is distinguished from other ailments, or in what respects it may differ from the same ailment in other people in similar circumstances. Thus a *diagnosis* is made by the art of converting *symptoms* into *signs* of disease.

But the physician at the same time generally carries his mental exertion a little further. He tries to arrive at a just estimate of the probable result or *event* of the malady, and so makes up his mind,—(4.) As to whether the illness will terminate in the death of the patient, in permanent organic mischief of greater or less extent, in persistent impairment of

the general health (*cachexiæ*), or in complete recovery. As in Politics, so in the science of Medicine: the Politician and the Physician have each to deal with the future, as well as with the present. Both endeavour to *forecast* events; and thus, in the practice of Medicine, we are said to make or give a *prognosis*. (5.) The Physician must be able also to appreciate with reasonable rapidity those *symptoms* which are peculiar, and to recognize them when associated together as the *signs* of particular or definite morbid states. Such *symptoms* are then said to furnish *pathognomonic signs* of disease. (6.) The Physician must further discriminate, and try to put a fair and just value or interpretation upon, those symptoms which are only experienced by the sensations (subjective) of the patient himself, as contrasted with those which may be seen or appreciated by others—such as objective phenomena or physical signs.

The interpretation of symptoms can only be successful after a close observation of the patient—often prolonged, and repeated for more complete investigation—so as to connect the results arrived at with his previous history. The utmost logical acumen is required for the due interpretation of symptoms. The individual value of each ought to be duly weighed; one symptom must be compared with another, and each with all; while the liability to variation of a similar symptom in different cases of a like kind must not be forgotten; and the occasional absence of the usual pathognomonic signs may be sometimes calculated upon. Thus only can the nature of a disease be clearly determined—its severity and dangers fully appreciated—its treatment indicated, and the probability of recovery foretold.

A close observation of general symptoms, in all their details, is absolutely necessary; and the investigation is aided practically by the improved instruments of the present day, and better methods of examination. Above all things, *methodical examination* is essential for the student, if he would acquire the habit of carefully and accurately examining the nature of the cases of disease with which he will have to deal. Patients must be examined methodically, in order that the symptoms of disease may be correctly interpreted, and that nothing be overlooked or neglected. Directions have been given by many authorities for acquiring and habitually following a definite system of examining patients, as

to what are the essential data to be obtained and recorded in case-taking; and although, as Dr. Acland justly remarks, a skilful practitioner can learn the truth of most cases in any order, or in no order, yet it is highly desirable that a regular order should be followed by learners; and all cases observed by the student should be methodically entered in a note-book for the purpose. This habit will thus eventually become a necessity, and will be found most useful in after-life, and especially in consulting practice.

The following works are recommended for study, and as guides for acquiring the best methods of observing and recording cases:—(1.) *A Manual of Medical Diagnosis*, by A. W. Barclay, M.D.; (2.) *A Handbook of Hospital Practice; or, an Introduction to the Practical Study of Medicine at the Bedside*, by Robert D. Lyons, M.B., Professor of Medicine in the Catholic University of Ireland; (3.) *An Introduction to Clinical Medicine*, by John Hughes Bennett, M.D., senior Professor of Clinical Medicine in the University of Edinburgh; (4.) “Suggestions for taking Cases,” by Dr. Beale, *Archives of Medicine*, vol. iii., p. 47.

CHAPTER VI.

MORBID ANATOMY AND PATHOLOGICAL HISTOLOGY: THE SPECIAL MEANS AND INSTRUMENTS BY WHICH THE NATURE OF DISEASES MAY BE INVESTIGATED.

MORBID or, as it is also sometimes called, PATHOLOGICAL ANATOMY is that department of medical science which treats of the changes produced by disease in the solids and fluids of the body; while MORBID or PATHOLOGICAL HISTOLOGY treats of the origin, development, growth, and decay of the new products or new formations which are the elementary constituents of structural or organic lesions. The anatomy of diseased parts stands in the same relation to the development of morbid phenomena and conditions of disease that the anatomy of healthy structures and the histology of the textures do to the natural functions and process of development, growth, and nutrition in the healthy body.

The vestiges left by the prolonged existence of a morbid state, whether in the body of man or of the lower animals, have always claimed from the physician a large share of attention; and in proportion as the knowledge of healthy anatomy and physiology has become extended and prosecuted in all its bearings, so has pathological science been extended, and morbid anatomy has gradually but steadily acquired an important and prominent position among those branches of study on which Medicine rests its claims as a science.

MORBID ANATOMY is a department of medical science which has gradually grown out of the accumulated experience and observation of ages; but PATHOLOGICAL HISTOLOGY, as a science, is of modern origin. It is but yet in process of development, although its foundations may be traced in the works of the earliest medical writers of antiquity. All of them refer to changes which they *merely supposed* had taken place in the internal organs; and they were doubtless led to this assumption by observing the connection that existed between structural lesions of the external parts and their accompanying symptoms. Hippocrates describes the deposit of tubercles in the lungs, the symptoms occasioned by them in a crude state, and those which attend their softening and discharge.

The science of MORBID ANATOMY is a record of facts. In its relation to the progress of Medicine it is a living record—a history whose pages must be ever open to receive the observations which are constantly being made by those engaged in pathological pursuits—a record from which one may ascertain at any time the conditions under which morbid changes or new formations in the body have taken place. The pages of this history show that at the present day the department of pathology is in a transition state; and the position of Medicine, as a science, must eventually result from a re-arrangement of the innumerable details which the sciences of morbid anatomy and histology may disclose and unfold. It is necessary, therefore, and often advantageous, to look back upon the past, and see what has already been done, so that its venerable facts may not be lost sight of, but grouped in series with the extensively verified experiments and observations of the present day. In so doing, if we pause and contemplate the steps which have been taken to arrive at our present position, such a contemplation may stimulate the

youthful student to the noblest exertions of his intellect, as he cannot fail, with extensive study, to see before him, and on every side, much unlaboured but productive soil. Such a retrospect will at the same time have the effect of placing in a prominent aspect the varied influences which morbid anatomy has had on the science of Medicine, the conditions under which it has flourished, and the legitimate objects of its investigations.

The art of printing had not been long invented when books on morbid anatomy began to issue from the press; and although the early period of the fifteenth century has left little enduring literature of any kind (but has been mainly distinguished by the number of colleges then founded), yet about this time pathological anatomy in the medical school of Florence shows the earliest evidences of an existence.

The facilities for study which the art of printing introduced soon stirred up ardent students; and the sixteenth and seventeenth centuries produced much that will ever remain famous in the annals of medical science. Eustachius, Tulpius, Ruysch, Harvey, Malpighii, and Leuwenhoeck are names familiar as household words to the student of medicine. The earlier attempts of this period to form a system of pathological anatomy is characterized by abortive endeavours to explain all results upon some exclusive and general principle. A spirit of speculation marks the character of the age. The men of that time had observed but few facts; and on these facts they preferred to speculate and dogmatize, rather than prosecute the further interpretation of nature, or record more observations. Accordingly, theories in abundance successively led captive the minds of the medical world, and, disappearing one after the other, demonstrated the unstable nature on which the science of Medicine had been placed. The leader of each sect founded his so-called school or system, all of them distinguished by a due amount of arrogance and contempt for predecessors and contemporaries—a feeling unhappily not yet quite extinct. The "*vital agency*," the "*influence of the humors*," and of the "*solid organs*," have each been considered by turns as the only orthodox belief; and each has had their school and sect respectively designated as the *Vitalists*, the *Humoralists*, and the *Solidists*. The theories of Galen, of Paracelsus, and others, have all been famous in their time, but are

now unheard of, and almost unknown. The same fate awaits the false theories and absurd conceits of more recent origin, although, as in the case of Stahl, Cullen, Brown, and Broussais, they have had a wide prevalence in the schools of Europe, and made impressions on the sentiments of the profession which yet influence their modes of practice and the reasons of their belief. Broussaisism, Hahnemannism, and some other systems, "the fruits of a luxuriant fancy and of few facts," must all descend, as others have done, the same inevitable slope to oblivion; but the vast collection of facts which the founders and followers of such systems eventually accumulate and bring to notice, remain unchangeable, and will continue to recur in the daily experience of our profession, just as they appeared to the venerable fathers of medicine centuries before the Christian era. The practice of medicine, as based upon rational principles and a knowledge of the nature of diseases, has oscillated through all these systems and theories, and the science of morbid anatomy has been marked throughout by unmistakable periods of *progress*, of *stationary existence*, or even of *retrogression*, according as one or other exclusive system had the ascendancy, or as each principle of practice challenged for itself a supreme importance.

The modern doctrines relative to the nature of diseases and the practice of Medicine may be said to be guided by the dictates of *Physiology*, and what is known regarding the development of the human body. Ordinary dissections alone, or *post-mortem* examinations of the body, have long since ceased to furnish us with facts before unknown; and new modes of extending observation and research, by taking advantage of every physical aid to the senses, are diligently looked for by the modern anatomist, physiologist, and physician; and the means and instruments which advance the science of physiology are well able to advance our knowledge regarding the nature of disease-processes.

A belief is now rapidly gaining ground, and acquiring a hold on the popular mind, that advances in the science of medicine in future years will be mainly due to a *better appreciation of the causes of disease*; and just in proportion as our knowledge of physiology and pathology becomes more exact and extended, so will the *causes of disease* be appreciated, and the *occurrence of disease* on a large scale prevented. An amiable and large-minded physician, Sir John Forbes, who but recently has taken his place

amongst the "Great ones of the Past," emphatically recorded the observation more than fifteen years ago, that "here the surest and most glorious triumphs of medical science are achieving, and are to be achieved." He himself lived to see great and good results; to see improvements in social and sanitary matters which continue to be realized, and whose rapid progress is characteristic of the present period. Within the last half-century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and often injurious in the first instance, the systematic sewerage of towns and draining of land have become of the first importance. Land has thus, in not a few instances, doubled its value. Town-sewering, with other social regulations, have contributed to prolong human life from 5 to 50 per cent. as compared with previous rates in the same district. Agues and typhoid fevers are reduced in the frequency of their occurrence. Since 1840 an annual mortality in English towns of 44 in 1,000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15. Not less remarkable reductions have taken place in the mortality and loss of strength in the army and navy; so that generally it may be said that human life has now more value in England than in any other country in the world—a result entirely due to better sanitary arrangements (Rawlinson "On Sewering of Towns," *Soc. of Arts Journal*, vol. x., p. 276).

The political economist cannot now, therefore, regard MEDICINE in any other light than as a productive art; and the labours of the Physician, whether in civil or in military life, cannot be regarded as unproductive labour.

But the science of Physiology (on which much of our sanitary improvements are based) has immeasurably outstripped the science of Pathology in the comprehensiveness of its views and in the value of its results; while Pathology, in its turn again, has always been, and ought to be, in advance of Therapeutics. The best physiologists have distinctly recognized that the basis of their science must include not only a knowledge of animals below man, but a knowledge of the entire vegetable kingdom. Without such an extensive survey of the whole realm of organic nature, we cannot possibly understand human physiology, and far less comparative physiology. The science of Pathology, therefore (whose aim is to expound the *nature of*

diseases), must be, *à fortiori*, very far behind. The diseases of the lower animals, for instance, rarely form any part of the study of the student of Medicine. The diseases of plants are almost entirely neglected. Yet it is clear that until all these have been studied, and some steps taken to generalize these results, every conclusion in pathology regarding the nature of diseases must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the causes of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food. (See chapters on Zymotic diseases.)

To physiology, therefore, in its most comprehensive sense, and to a knowledge of the natural and normal development of animal and vegetable beings, we must look for future progress in pathology; while the means and the instruments which advance physiology will simultaneously advance our knowledge regarding the *nature of diseases*,—a sound knowledge of which can alone enable us to “*appreciate their causes*,” and so arrange measures for the *prevention of many of them*, based on the great truths of science.

Organic chemistry, the microscope, the ophthalmoscope, and such-like instruments, have opened up new fields of labour, which are being diligently cultivated; and while alterations in the ultimate tissues and organs are more especially attended to, the first beginnings of disease, the development of new formations, and the examination of excretions, claim a large share of attention.

Histology, or the study of the development and arrangement of the tissues in the formation of normal and healthy organs, is characteristic of the anatomical investigations of the present day; while the histology of morbid products and chemico-physiological investigation into the nature of morbid changes is characteristic of the pursuits of the science of modern PATHOLOGICAL ANATOMY.

It is also a significant fact that now, in the nineteenth century, some of the leading doctrines of the *humoral* pathology which prevailed in the seventeenth are again revived. The experience and learning of that erudite period are now being made available for modern uses. By the improved means, instruments, and

methods of research of modern times, important truths may be sifted from the errors and theories with which they are mixed up in the ancient chronicles of medical science; and when we get analogous conditions of disease with which the phenomena described by the ancients may be compared, "not a few of the apparently modern beliefs are daily found to have a time-honoured reputation unappreciated before."

The chemist and the histologist now combine their researches, and work hand in hand; and we regard them as the most inquisitive anatomists of the time. They lend assistance of the most important kind in working out the foundation of our knowledge regarding the nature of diseases, the details of which can only be made more certain and perfect by taking advantage of every kind of scientific knowledge which can be brought to bear upon medical research, and more especially,—(1.) By physical aids to the senses, extending our means for the actual inspection and appreciation of phenomena. The use of the stethoscope, of the microscope, thermometer, ophthalmoscope, laryngoscope, and specula of various kinds, aided by a careful study of the writings and labours of the men who have more particularly devoted their attention to observations by such means, may be quoted as examples (LAENNEC, LOUIS, WALSHE, STOKES, HOPE, BENNETT, QUEKETT, VIRCHOW, WUNDERLICH, TRAUBE, VOGEL, BEALE, GRAEFE, CZERMAK, and others). (2.) By the knowledge (gradually being made more extensive) of the textures, organs, and functions of the body, whose normal exercise constitutes a healthy existence (LONGET, MULLER, SHARPEY, VALENTIN, ALLEN THOMSON, CARPENTER, KIRKES, PAGET, KÖLLIKER). (3.) By an intimate knowledge of the normal development of the human textures, as well as those of plants and animals from the fecundated ovum (BISCHOFF, COSTA, ALLEN THOMSON, HUXLEY, NEWPORT, and KÖLLIKER). (4.) Besides these kinds of investigations, the science of practical medicine has been, and is being, advanced by operations and experiments upon the internal organs of living animals, opprobriously termed *vivisections*. At some of our great schools of medicine such investigations are now being actively but judiciously prosecuted and taught; as by Bernard, in Paris, Drs. G. Harley, Brown-Séquard, and Pavy, in London.

Successful inquiries into Pathology, or the nature of diseases,

cannot be said to have commenced till the middle of the eighteenth century, when the great work of Morgagni issued from the press. It was the work of his lifetime. In the eightieth year of his age, and not till then, did he consider himself warranted to publish his observations, *De Sedibus et Causis Morborum* (1761); a work whose material and circumstances of publication read us the practical lesson, that the more frequently a disease occurs, the more necessary it is that its phenomena should be carefully investigated. And when we think of the prudent reserve, the anxious and the conscientious delay exhibited by Harvey, Morgagni, and Jenner, in the publication of their respective researches, we cannot but contrast the circumstances with those under which the exuberance of medical publications are now given to the world. Morgagni modified and corrected many of the views entertained and promulgated by his predecessors; and the study of the nature of diseases was carried into the commencement of the present century by CULLEN, DE HAEN, WILLIAM and JOHN HUNTER, PORTAL, and BICHAT.

The knowledge of the physician regarding the nature of disease-processes may now be observed to have advanced simultaneously with that of *general anatomy*; and when the component parts of an organ, and of the human body, came to be distinguished, it was soon observed that membranes and tissues might be individually diseased while neighbouring membranes and tissues remained untouched. Bichat's idea, therefore, of decomposing the animal body into its elementary parts must be regarded as the foundation of modern special pathology; and while he pointed out the necessity of studying diseases with reference to the different tissues as separately and specially affected, it has been since shown, in a remarkable manner, how general anatomy, deduced from physical properties of parts and crude observation, may coincide with more minute investigations of a chemical and microscopical kind. The membranes and tissues composing the organs of the body, roughly torn asunder by Bichat, are now themselves being daily subjected to a more inquisitive analysis of an anatomical and chemical nature, which unravels them into still more minute histological elements.

Although, therefore, Bichat entertained the view that each tissue had its own *diathesis*, it is to Cullen and the Hunters in this country more especially that the application of the dis-

inction of tissues was made to illustrate the nature of disease-processes.

Cullen's descriptions of diseases are descriptions of groups of phenomena which comprise complex morbid states.

The written labours of the Hunters form but a small part of the memorials of what they did to elucidate the nature of diseases, and it is only those who have had the opportunity of carefully examining their museums, preserved in London and in Glasgow, that are able to form any conception of the comprehensive nature of their labours, or to assign to them a proper place among those who have successfully advanced the science of Medicine. They hold a position at least one hundred years in advance of the age in which they lived. Bichat, Cullen, and the Hunters, in their respective countries, have thus reciprocally influenced and advanced the progress of our knowledge regarding the nature of diseases. And although it was reserved for Bichat to complete a more perfect system of general anatomy, it must not be forgotten that Dr. Carmichael Smith, in 1790, applied his knowledge of textural anatomy to elucidate the nature of disease-processes; and that Pinel, after him, in his *Nosographie Philosophique*, made the distinction between the membranous and other animal structures the foundation of his pathology. The classic work of Baillie (his *Morbid Anatomy*), published in 1793, closed the labours of the past century.

If now we look to the tendency of the studies and researches of those men just named, including Bichat, we shall find the truth gradually being more fully appreciated, that it was necessary to study alterations of structure so as to connect morbid changes with the symptoms of diseases during life, and with the operations of ascertained causes of morbid action. The nature of the morbid changes were now observed to be more apparent in the progress of external diseases; and therefore surgical experience was brought to bear upon the elucidation of internal disease-processes.

Thus the progress of Morbid Anatomy is, in a great measure, a record of the history of Medicine; and we can trace the science of special morbid anatomy, giving a character to the various systems of the healing art which have prevailed from time to time.

All the writers up to the time of Bichat, Laennec, and Aber-

crombie were pure morbid anatomists, who did not connect the effects of disease with their causes, and who recognized the changes of disease as important only in proportion to their magnitude as apparent to the senses. They are therefore regarded as pure solidists, whose researches doubtless contributed much towards a correct knowledge of the changes in the organs of the body, while the condition of the fluids was neglected, as well as the relations of the texture, organs, and fluids, in the combined exercise of their functions. Simple functional disturbances were thus wholly overlooked, and the constitutional connection of local affections entirely lost sight of.

The cotemporaneous surgery of the period previous to Bichat was marked by its unwillingness to recognize anything but material facts, mechanical processes and contrivances. The surgeons of those days desired to know nothing but anatomy and mechanics; and, accordingly, it may be recognized as the period of pure anatomical and mechanical surgery, distinguished by the writings of men whose works bear ample testimony that the surgery of the period was founded on exact and even minute anatomical knowledge. No allusion is made, however, by them to medicine,—they make no application of physiological truths, and they encourage no therapeutic tendency apart from mechanical or instrumental interference.

The purely solidist, as well as the purely humoral principles by which the nature of diseases have been explained, may be said to have died a natural death long ago; but, as already noticed, the remembrance of what is valuable in the results of both are preserved in modern pathology, which takes its stand upon anatomical and physiological facts, connected by simple methods of inductive observation with the symptoms and signs of disease as seen and expounded to the student by the distinguished professors of Clinical Medicine at most of our celebrated schools, where Clinical Medicine is taught.

In this field of instruction it seems invidious to mention here the names of men still living. For their own sakes, as well as for science, may they be long deprived of being thus honourably and respectfully mentioned. As teachers, they are in our own country familiar to every student. As recorders of what they observe at the bedside and after death, they are not less celebrated abroad than appreciated at home.

Tested by extensive clinical observations, the character of the present period in the history of Practical Medicine is one of *probation* as well as of *progress*, marked by a close inductive examination of past generalization and classification of facts, however remotely connected, which illustrate the nature of diseases and their treatment.

Side by side, since 1816 and 1819, the microscope and the stethoscope, under the influence of such men, have advanced our knowledge of the nature of diseases with a regular and accelerated velocity; but they have only done so as assistants and in subordination to laws and facts whose knowledge we have acquired by a close observation of general symptoms, of which such instruments have never been intended to take precedence. They have never accomplished, nor can they ever accomplish, useful practical results, to the exclusion of such other methods of observation as have just been noticed. We are not to confound *relative* smallness with *absolute* simplicity, and believe that because a simple organic cell is a small object—because we can see around it, through it, and on every side of it—the functions and conditions of its existence are less *complex* or less obscure on that account than are those of a more complex organ, or the functions of a living body.

We are not to suppose that because the stethoscope enables us to detect a mitral murmur, or a crepitation in a lung, we are justified at once in adopting one, and only one, method of treatment. It is this exclusive use of instruments, to the disregard of general symptoms and signs of disease, derived from close observation and knowledge of the living functions, which leads to the repudiation of the use of such instruments by the sagacious and experienced physician, who sees the numerous errors not unfrequently committed by his younger brethren, who trust too exclusively to these instruments in the diagnosis of disease.

Like the stethoscope, the microscope has been unjustly and unnecessarily burdened with labour, and has been equally unjustly blamed, and brought into unmerited discredit, when it has failed to elucidate the nature or even presence of a morbid state, the existence of which could not be doubted, but which the sense of sight could not appreciate, even when presented in small quantities greatly magnified. In such instances the microscope has been applied to uses which it is not the nature or province

of the instrument to detect. The gravimeter or hydrostatic balance, the microscope, the stethoscope, the ophthalmoscope, the laryngoscope, the pleximeter, and the thermometer, are merely instruments of pathological inquiry, each one adapted for the determination of particular classes of facts. They can only elucidate disease when they are brought to bear upon physical properties, the nature of which they are able to appreciate; and it is only from their *combined and appropriate* use, in connection with a history of the general signs and symptoms in each particular case, that our knowledge of the nature of diseases will be advanced.

The industrious employment of these aids to diagnosis, and an intimate acquaintance with the results, are attended with this further advantage, that such practice and knowledge enable their possessor to appreciate the general symptoms of disease with infinitely greater certainty than heretofore. This is the usual consequence of training in all exact methods of observation. The thorough study of these aids to the senses in appreciating disease leads directly to the possibility of dispensing with them in many instances. By means of auscultation and percussion, for example, our attention has been drawn to numerous conditions of the thorax, which enable us to make the diagnosis at the first glance, which hitherto was not possible; because the conditions for diagnosis could never have been recognized without such physical aid to the senses as that derived from auscultation and percussion. In many cases, from the mere inspection of a patient, a well-instructed clinical student may decide upon the existence of *pleurisy*, *pneumothorax*, *emphysema*, or *pulmonary tubercle*. The initiated are thus frequently enabled to dispense with percussion and auscultation; but if they had never acquired the practical knowledge of the subject—if they had never examined numerous patients by means of these physical aids to diagnosis—and so learned thus to determine with great exactness the significance of the various forms and movements exhibited by the thorax, they would never have been able to appreciate their significance. So, also, the physician well instructed in the use of the thermometer may, in hundreds of cases, without its aid, draw conclusions with great certainty, incomprehensible to others not so instructed; but if, led away by this skilfulness, he is induced to dispense with *exact* thermometrical control, he may soon fall into gross errors. So it

is with the ophthalmoscope, specula, and all other more or less exact physical aids to diagnosis. Let them be in constant and appropriate use, but the results must always be taken and compared in connection with other general symptoms of disease.

In all the temperate regions of the world, histology, as applied to morbid products, has been cultivated, and has advanced our knowledge regarding disease ever since 1838. In warmer latitudes our knowledge of practical Medicine has been advanced by extensive observations on physical climate, medical topography, and by organic chemical analysis applied to obtain therapeutic agents from the vegetable world. Those may be said to be the characteristics of the researches of our own country, Germany, France, and America, as contrasted with the nature of the observations mostly prosecuted in India.

No exclusive doctrine will now stand the test of well-directed pathological inquiry, the main object of which is to connect all organic changes (lesions) and functional derangements with their symptoms and causes, with the view of applying rational remedies and prophylactics. The too exclusive study of pure organic pathology and morbid anatomy leads to no distinction between the signs and causes of disease; and the obvious tendency of such exclusive study is to exaggerate the importance of the principles it may establish, to hold out no hopes of cure, and to undervalue the power of remedies and remedial measures. To obviate this tendency it is necessary to have recourse to inductive reasoning, so as to connect all the morbid changes seen or appreciated after death with the signs and symptoms of disease observed during life. Thus it is that links in the chain of disease-processes which, from a one-sided or exclusive view, appear isolated and localized, are really found to be connected with each other. It may be, also, that they are connected with a long but intelligible series of processes developed during life through the metamorphosis of tissue, and going on in apparent health, or in an obviously morbid exercise of function. The constitutional origin of many local diseases, otherwise inexplicable, then becomes apparent.

Among the more eminent exponents of this rational school of pathology, who at an early period in this country discerned and appreciated such doctrines, we find the names of ALLEN, GOLDING BIRD, SIR ROBERT CARSWELL, GREGORY, HOPE, HODG-

KIN, MARSHALL HALL, PROUT, WILLIAM STARK, JOHN THOMSON, TWEEDY TODD, and many others, who, although now no more, have left behind them imperishable evidence of their labours. The younger pathologists of the present day, whose name is *Legion*, follow in the footsteps of these men, extending the fields of observation and the boundaries of the science of Medicine. By them the importance of morbid anatomy is sufficiently appreciated, and its province distinctly defined and limited as follows, namely:—(1.) To detect the changes which have taken place during the course of diseases in the structure of tissues and organs of the body; (2.) To demonstrate the exact seat of local alterations established during the progress of disease.

The investigation and elucidation of the *nature, course, and causes* of those changes, constitute the prominent objects of the science of pathology. By the aid of morbid anatomy and clinical observation during life, pathology seeks to establish the relations of the changes which lead to the lesions, and so to connect the general progress of disease with its symptoms and signs.

MORBID ANATOMY goes beyond its province when it attempts to point out the nature of the proximate cause of disease. It is only by the application of inductive reasoning that the connections of causes and morbid effects can be shown, and such constitutes the main object, and is the highest aim of the science of PATHOLOGY.

The *morbid anatomist* finds a lesion or change for what ought to be the natural structure, appearance, or condition of a part. The *pathologist* seeks to connect such lesions with signs and symptoms during life, that the *practical physician* may suggest a remedy to the disease, and that the *nosologist* may give it a name, distinguishing characters, and a place in his classification.

CHAPTER VII.

THE ELEMENTARY CONSTITUENTS OF LESIONS AS SHOWN BY
MORBID ANATOMY AND OTHER MEANS OF RESEARCH.

WHERE the material effects of disease can be rendered obvious they are found to consist for the most part of,—

1. Morphological changes in the elementary textures of the body generally, and altered conditions of the fluids.

2. The presence of new formations foreign to the normal condition of an organ or system of organs.

3. Change in the position or form of some of the organs or parts of organs.

4. Deposits in or around the elementary parts of tissues, or changes of a degenerative or retrograde kind in them.

The object of prosecuting the anatomy of disease is, therefore, in the first instance, to institute a comparison between the known appearances or standard of health and an altered state of the parts. Such a comparison is, in the first instance, founded on an intimate knowledge of the doctrines stated at page 4.

Means and Instruments of Research.—To institute investigations such as those indicated at page 10, advantage must be taken of almost every branch of human knowledge. The methods of carrying on pathological research are therefore very varied, but may be shortly enumerated under the following heads:—

1. The opening of dead bodies, to ascertain the condition of their organs and tissues in all that relates to their structural, chemical, and physical properties (ROKITANSKY, HASSE, VIRCHOW).

2. Application of various instruments, such as the microscope, and of means to ascertain the absolute and specific weight of organs or parts, the relations, size, form, and colours of structures, and the like (QUEKETT, BENNETT, BEALE, PEACOCK, BOYD).

3. Application of chemical investigations to the diseased products (VOGEL, SIMON, DAY, LEBERT, GLUGE, BEALE, GARROD, CHRISTISON, PARKES, VIRCHOW, FRERICHS, GAIRDNER).

4. Application of statistics to determine various points of interest in reference to the nature, course, and complications of diseases (WM. FARR, GUY).

5. Means to preserve objects for further study by the microscope, or any other mode of examination (TULK, HENFREY, BEALE, QUEKETT, VAN DER KOLK, LOCKHART CLARKE).

6. Experiments instituted on living animals, and, in certain cases, on man, with the view of artificially producing a morbid condition. A careful study of such experiments by the previously mentioned means affords valuable information, for the causes in action are more under control than those which are spontaneously brought about by disease in the living body (BERNARD, HARLEY, PAVY, KUCHENMEISTER, ZENKER, and others).

The immediate object of such investigations is to obtain information regarding the material changes in the different parts of the body which accompany or produce morbid symptoms, and to connect these changes with symptoms and signs of disease during life. We thus learn how morbid products are formed at first and gradually perfected; and by combining these two kinds of knowledge we learn the relative connection of two orders of phenomena; namely, how the perverted properties, disordered actions, or altered structures give rise to perverted or impaired secretions; disordered and irregular motions; deranged, impeded, or interrupted functions. In other words, the "*order of invasion of disease-processes*" is learned from such investigations; and we are thereby taught how parts, once the seat of morbid change, return, by various processes of nutrition, growth, repair, or reproduction, to their normal condition.

The questions arising out of such investigations are, or ought to be, the first object of thought to the conscientious medical practitioner. It is his duty, from an attentive consideration of the signs and symptoms of disease, to form an idea, as accurate as possible, of the nature and extent of the morbid action or change which is going on, or which may be set up, in the tissues, organs, and fluids of the living body.

If, therefore, he does not avail himself of every means and instrument by which he can ascertain the existence of change in the dead body, and its alteration from some standard of health—if he does not embrace every opportunity of making *post-mortem* examinations—if he contents himself merely with observing signs or symptoms of disease, without witnessing the changes of structure, if any, which may give rise to them—he can have little conscious satisfaction in the study of Medicine as a science, or in the

practice of the healing art. In the words of Cruveilhier, he will, during his lifetime, "see many patients, but few diseases." Such a practitioner is not to be trusted.

Various Forms of the Constituent Elements of Disease.

The histologist has now clearly ascertained the various simple organic forms which compose the textures in their normal state, and the mode in which these textures are arranged and combined so as to form the organs and systems which carry on the healthy functions of the body. The pathologist has made out (although with less completeness), by the methods of observation and experiment already indicated, the various simple organic forms which constitute the elements of those material changes whose phenomena of growth, decay, and varied change are associated with the manifestations of disease. By classifying and arranging these forms we obtain more clear ideas of lesions; and we ascertain that the material morbid processes follow, in their development, a very definite order of change, not yet in all cases determined with absolute certainty.

An anatomical investigation of morbid parts, conducted with the aid of the microscope and other instruments of research, shows that the material of which their substance is made up is of very various structure, sometimes combined in forms of one kind throughout, and sometimes varied by the development and combination of many elementary forms, more or less solid, soft, or fluid.

An analysis of the morbid material, carried as far as scientific means at present enable us, shows that the elementary conditions in which morbid products are found may be described as follows :—

1. Fluid matter and hyaline substance, more or less soft.
2. Simple elementary forms of the nature of deposits, sometimes of a mineral or inorganic character; *e. g.*, (*a.*) amorphous granules; (*b.*) crystalline structures in a granular state.
3. Simple, but organized products capable of growth; *e. g.*, (*a.*) granules; (*b.*) compound corpuscles; (*c.*) simple cells; (*d.*) fibres.

The various appearances and conditions which these simple forms may assume in disease, as well as the functional states with which they are frequently associated, lead to a further

enumeration and classification of morbid elementary products, as well as of more complex disease-processes, as below:—

A.—MORBID ELEMENTARY PRODUCTS.

I. EXUDATIONS MORE OR LESS SOFT, SEMI-FLUID, OR FLUID, AND FORMED OF,—

a. Germinal plastic and formed material, which has sometimes been called *blastema*, *coagulable lymph*, *false membrane*, or *fibrine*, as seen adhering to free surfaces.

b. Aqueous matter, as seen in the morbid state termed "*dropsy*," and "*œdema*" of parts.

c. Gaseous exudations, as seen in the various forms of *pneumosis*.

II. EXUDATIONS MORE OR LESS CONSOLIDATED, AND CONSISTING OF,—

a. Molecular or granular material, from the 800th of a line to an immeasurably small size, and consisting chiefly of the simple forms of,—

(1.) Fatty molecules or granules.

(2.) Forms of an organic kind capable of growth, and invariably taking origin from a pre-existing structure.

(3.) Deposits of an inorganic kind, generally calcareous salts.

(4.) Pigment granules.

b. Coagulable compounds, resisting the action of most reagents, such as are seen in the elements of tubercle, scrofula, oleo-albuminous formations.

c. Exudations of a transitional nature, organized, which are capable of growth, which may become vascular, which grow from pre-existing structures, and which are composed of,—

(1.) Consolidated homogeneous material passing to

(2.) A fibrilloid arrangement of the molecular or granular particles composing connective substance, and a subsequent formation of fibres in it or from it.

(3.) The formation of pyoid cells, and fibro-plastic or connective tissue cells, passing into fusiform cells and fibres as the material becomes consolidated.

(4.) The formation or exudation of fluid matter holding pus, or other more compound cells.

III. GROWTHS AND EXUDATIONS OF A MORE OR LESS SPECIFIC KIND.

- a. Lymph of small-pox and cow-pox.
- b. Matter of glanders, of malignant pustule, and of the plague.
- c. Fluid of infecting chancre, and of some forms of secondary syphilitic lesions.
- d. Material of tubercle and scrofula. (?)
- e. Material of cancer.
- f. The growth in Peyer's glands during typhoid fever.
- g. The growth in Peyer's glands in cases of cholera.
- h. Melanotic or pigmentary germs.

IV. MATERIAL OF A COMPLEX KIND.

- a. Media of repair and reproduction of injured or lost parts—substance of granulations and cicatrices.
- b. Hypertrophy of parts.
- c. Tumors, $\left\{ \begin{array}{l} \text{innocent.} \\ \text{malignant.} \end{array} \right.$
- d. Concretions.

V. PARASITIC FORMATIONS.

B.—COMPLEX VITAL PROCESSES WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE.

- 1. *Fever*—the febrile state—*Pyrexia*.
- 2. *Inflammation*.
- 3. *Irritation*.
- 4. *Congestion*.
- 5. *Depression*.
- 6. *Atrophy*.
- 7. *Degeneration*.

Such a classification as the above is merely intended to bring before the student at a glance the variety of morbid material which is concerned in the expression of many of those phenomena seen in the course of disease, the distinctions made being mainly based on structural analysis.

While it is more properly the province of the anatomist to describe the MORBID ELEMENTARY PRODUCTS, it is the COMPLEX VITAL PROCESSES, WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE, with which the Physician has more immediately to deal; and some of these complex states especially require notice here; namely, *Fever and Inflammation* and some forms of *Degeneration*.

CHAPTER VIII.

COMPLEX MORBID STATES.

SECTION I.—FEVER—*Pyrexia*.

Definition.—*A complex morbid state which accompanies many diseases as part of their phenomena, more or less constantly and regularly, but variously modified by the specific nature of the disease which it accompanies. It essentially consists in elevation of temperature, which must arise from an increased tissue-change and have its immediate cause in alterations of the nervous system (VIRCHOW, PARKES).*

Pathology of Fever and Phenomena which constitute the Febrile state.—In describing the nature of fever, the following statements are principally compiled from the Gulstonian Lectures of Dr. Parkes, delivered before the College of Physicians in 1855, and from a review by Dr. Jenner, “On the Proximate Cause of Fever,” in *The British and Foreign Medico-Chirurgical Review* for 1856. Knowing how difficult it is to convey an orthodox account of the nature of fever; fully impressed with the great importance of the subject; and believing, as Dr. Jenner has expressed, “that so consistent a theory of the nature of fever, and one so largely supported by facts, has not been placed before the profession as that developed by Dr. Parkes, I only hope I may be able to do it justice in the attempt to lay it before the student of medicine in the following form. In the eloquent language of Dr. Parkes, ‘I shall have to allude to inexplicable phenomena, to vast spaces still unfilled by solid facts, to spots unknown to observation, and to regions lighted only by the dim and treacherous ray of speculation.’”

The practical object aimed at in the exposition about to be given, is to fix the scientific principles which ought to guide clinical investigation in determining the *Natural History* of fevers generally; and especially to define the differences which subsist among specific fevers; and so aid in determining the conditions under which fevers are generated or propagated—their development, course, or progress, and their defervescence.

“A hot skin, a quick pulse, intense thirst, scanty and high-

coloured urine," are phenomena common to many diseases; and when they are present it is said that the patient is *feverish*, or suffers from *fever* or *pyrexia*. There are some diseases in which such symptoms constitute the prominent, and almost the only appreciable phenomena, and which run a more or less definite course, without the necessary development of any constant local lesion. Such diseases have been emphatically termed "fevers," or sometimes *specific*, *primary*, or *idiopathic fevers*. When diseases marked by local lesions—such, for instance, as the local inflammations—are attended by the symptoms just stated, then the *pyrexia*, *fever*, or *feverish* symptoms which attend them, are said to be *secondary* or *symptomatic*; and the physician is accustomed, when he deals with such cases, to abstract the symptoms of fever from the other symptoms proper to the special affection. In other words, he prescribes for, and tries to cure the special affection, and not the *fever*, because he knows that when he has subdued the local disease the *fever* will subside. Not so, however, with the fever of a specific disease like *small-pox*, *typhus*, or *typhoid fever*: the physician cannot cure such a fever; but he may guide its course, by judicious management, as an experienced pilot may guide a ship and preserve it through a storm.

It is to the nature of *fever* considered in its abstract relations that the attention of the student is here directed, and not to any particular fever, such as *ague*, *typhus fever*, or the like. It is to *fever* in general that the following observations apply. It is to the *pyrexial symptoms* which are common to many diseases, such as to *small-pox*, *scarlatina*, *measles*, *typhus*, *ague*, *pneumonia*, *nephritis*, *meningitis*, and which, "like shadows to substance, are necessary to the very existence of such diseases, but yet are not, *per se*, any one of these diseases."

Galen defined fever as a preternatural heat—" *Calor præternaturam*." Subsequently many other additional clauses were added to this definition, such as "quick pulse," "turbid urine," and the like; but still the improved definition would not meet the requirements of every case; and now it is fully recognized that of all the clauses and phrases in the usual definitions of fever, "preternatural heat" is the only one whose accuracy is unimpeachable. In all cases, therefore, where fever is present, there are two points to be determined; namely,—(1.) *The amount of the preternatural heat* by accurate measurement; (2.) *The*

amount of the tissue-change, as represented by an estimation of the relative amount of all the excreta to the body weight.

It is the exact sequence of phenomena we desire to know in every case where pyrexia is present, as well as the meaning and co-relation of the phenomena: and usually symptoms sufficiently characteristic become developed and superadded to the febrile phenomena, by which the physician is able to define the specific nature of the disease or fever as a whole, and to say of this case or of that, "It is a *typhoid fever*," or "It is an *ague*," or "It is a *rheumatic fever*," or "a *pneumonia*," or "a *dysentery*," or any other form of illness where pyrexia is present, which we are able clinically to recognize. It is not very long since we were able to do this. Up till within a comparatively short time ago, the classification and diagnosis of "Fevers" was not such as to distinguish and separate their varied forms and varieties from each other. "Common continued fever," for example, was a comprehensive name which included many very different types of fever; and no means of observation have been of late so exactly discriminating, so as to distinguish one form of disease from another where fever co-exists, as accurate observations on the temperature of the patient, determined by the thermometer. In acknowledging this great fact, it is important to observe that the absence of such exact observation, and the trusting to general signs alone, have hitherto led to great confusion—a confusion which has been most unjustifiably and unfortunately increased by a pernicious system—becoming too common—of naming "Fevers" from the place or locality where supposed varieties of fever have prevailed as epidemics; or by the use of local or provincial native names. For example, the Walcheren Fever, Levant Fever, Mediterranean Fever, Crimean Fever, Bulam Fever, African Fever, Fernando Po Fever, Lisbon Fever, Bengal Fever, Pucca Fever, Gall-sickness of the Netherlands, Hong Kong Fever, and other names not less barbarous, may be quoted. Except as matter of history, and as beacons to warn us from the danger to science, let these and such-like names be consigned to oblivion. With the exact means at the disposal of the physician as aids to diagnosis (and which are about to be described), every variety of illness where fever takes a part may be accurately distinguished, its type recognized, and its place fixed in nosology; or if it should be anomalous, its exact departure from the type may be not less accurately defined and described.

The phenomena which thus call for special investigation are those which are strictly related to the development and progress of the febrile state. They ought to be determined by clinical observation in all cases of disease where fever may be present. The facts to be ascertained are not less significant of the abatement, subsidence, or "*defervescence*" of the febrile state than of the advent of local lesions. The term "*defervescence*," in fever, is a comparatively new one in English pathology. It was first used by Professor Wunderlich, and subsequently adopted in this country by Dr. Parkes. It signifies the period during which the temperature of the fevered body is declining to its normal amount from that intense degree of heat attained in the state of accession of the febrile phenomena. This "*defervescence*" may be sudden, when it is regarded as a "*crisis*;" or it may be gradual, and is then described as a "*lysis*"—the "insensible resolution" of the older authors; or it may be partly sudden and then slow, when it may be described as "*wave-like*," with gradual and sometimes regular alternations of high and low temperature, as Dr. Parkes was the first to point out (*The Composition of the Urine in Health and Disease*, p. 270).

The Usefulness of the Thermometer at the Bedside in the Diagnosis of Pyrexia.—One hundred and ten years ago (1754), Antonius de Haen, the first teacher of clinical medicine in the Hospital of Vienna, impressed his pupils with the necessity of attending to the temperature of the body in disease, *as measured by a thermometer*, instead of being judged of merely by the hand. He showed that even in the cold stage of ague, with the teeth chattering and the body shivering, the temperature of the blood is rapidly rising, although the pallid skin may really be colder than usual—its supply of blood being diminished by the contraction of the blood-vessels. He first demonstrated with *measured accuracy* how much the heat of the body is augmented under the influence of the febrile state; and when the crude appliances and the rough instruments of a hundred years ago are compared with the delicacy and refinements of "the instruments of precision" of the present day, it may be of interest now to observe how the progress of knowledge and the powers of modern research have not suffered the valuable pathological lessons to be lost sight of which are to be learned from the clinical use of the thermometer, as de Haen taught a hundred years ago. When the hand of the

physician alone is used to judge of the temperature of a patient, or when the feelings of the patient are alone taken as a measure of his temperature, it can easily be understood how such kind of observation is extremely fallacious, doubtful, and unsatisfactory. The determination of the amount of heat in fever cases is stamped by a much more early appreciation of its importance and value than even since the time of De Haen; for ever since the days of Hippocrates, the Physician and the Surgeon have been in the habit of applying the hand to the skin of the patient, to appreciate the presence of abnormal heat; but the practical application of the thermometer in place of the hand, while it is obviously a more accurate method, has never come into general use, mainly on account of the difficulty of getting instruments sufficiently sensitive and trustworthy—instruments, in fact, of sufficient precision. The time and trouble required to work with crude and inefficient instruments soon brought them into disuse and discredit; but now the instruments required may be obtained so delicate and accurate, and the time they take to apply them is so insignificant, that the student of medicine and the physician have no excuse for neglecting to use them. When it is remembered, also, that Galen's definition of fever is still the one whose accuracy remains not only unimpeachable, but fully demonstrated, and now recognized; that it describes fever to consist in "a preternatural heat,"—it is obviously absolutely essential that medical men should be able to measure this heat, and so learn the significance of such increase of temperature in every case of disease where fever may be present. The careful physician *counts the pulse and the respirations* in all cases of illness; it is now not less incumbent upon him *to measure the heat*. By means of a delicate thermometer he has in every case of fever an accurate measurer of its amount; and the student of medicine, as one of the earliest clinical lessons in hospital wards, should be taught to look to the excreta, and to the various physiological conditions of the patient, for the products of the metamorphosis of tissue equivalent to the amount of heat in each disease.

For physicians the thermometry of disease is practically important from two points of view, inasmuch as,—(1.) *The continuous daily use of the thermometer greatly facilitates the clinical recognition of diseases.* It aids the busy practitioner in coming to certain and safe conclusions; and so relieves him of much anxiety of mind

in doubtful cases. (2.) *The use of the thermometer tends to elucidate the NATURAL HISTORY of all diseases where fever is present.*

It is proposed, therefore, to illustrate this subject under the following four heads:—

I. *The Instruments, Methods, and Practical Rules for Observing and Recording the Temperature of the Human Body in Diseases where FEVER may be present.*

Animal heat has been determined in two ways; namely, either by the ordinary mercurial thermometer, or by the thermo-electric apparatus. The latter is able to indicate fractions of a degree, and in this respect surpasses the powers of the most delicate mercurial thermometers. MM. Becquerel and Breschet employed such an apparatus to determine the temperature of internal parts. The apparatus consisted of two wires, of different metals, soldered together, and having their free ends brought into communication with a *thermo-electric* multiplier, having an index showing 10ths of a degree. The wires being passed through different parts of the body (like acupuncture needles) indicate the temperature of the tissues at the point of contact of the two metals. For example—passing the wires an inch and a half into the calf of the leg, the temperature was found to be 98° Fahr., while at the depth of a third of an inch it was only 94° Fahr., showing some cooling of the body towards the surface compared with the interior. The superficial fascia of the biceps was nearly 3° Fahr. lower than the temperature of the muscle itself. But notwithstanding the greater delicacy of the *thermo-electric apparatus*, a sensitive mercurial thermometer, finely graduated, is the only instrument of practical usefulness, as yet, for ordinary clinical purposes;—and for obvious reasons.

The Germans, who have been mainly instrumental in elucidating this subject, use thermometers which are graduated to Reaumur's or Centigrade scales—neither of which are in general use in this country.* These thermometers are cheap, and somewhat fragile, often differing also very much from each other. If German thermometers are used, it is therefore necessary to compare the thermometer used with a standard one, and note the differences *between every degree*. A thermometer is bad, and all but useless, if the differences between various degrees are unequal, but is quite serviceable if the same sum is to be added or subtracted for *each degree*. The price of such an instru-

* Mr. Griffin, of 119 Bunhill Row, has been able to import very good thermometers from Germany, graduated in Fahrenheit scale.

ment is moderate; therefore it need not be difficult nor expensive for a student to acquire a competent practical knowledge of "the thermometry of disease."

As it is necessary to have a good thermometer, with a uniform and correct scale, having a range from 88.2° to 110.7° Fahr., exhibiting also 5ths Fahr. of degrees, I have arranged, in a convenient wooden case (with the aid of Mr. Casella, the accurate and careful instrument-maker at 23 Hatton Gardens, London), two thermometers graduated in Fahrenheit degrees, especially for the use of medical men. Each box contains—

1. *An Ordinary but very Sensitive Thermometer*, fig. A, made with a curve, in order that its bulb may be the more easily and perfectly fitted into the axilla, while the stem being carried upwards, renders the reading *in situ* more easy.

2. *A Straight Thermometer*, fig. B, which, being a maximum self-registering one (known as "Phillip's maximum"), does not require to be read *in situ*, but may be removed from contact with the part, and read when convenient. Both thermometers are graduated up to at least 112° Fahr., and each degree is subdivided into *fifths*.

Directions for Use. I. *The Curved Thermometer*, Fig. A.—Its bulb must be well fitted into the arm-pit, being introduced below the fold of the skin covering the edge of the *pectoralis major* muscle, and so kept in close contact with the skin, completely covered and firmly surrounded by the soft parts. In very thin or very old persons this adjustment requires special care. The instrument must be retained *in situ* during a period of not less

Fig. A.

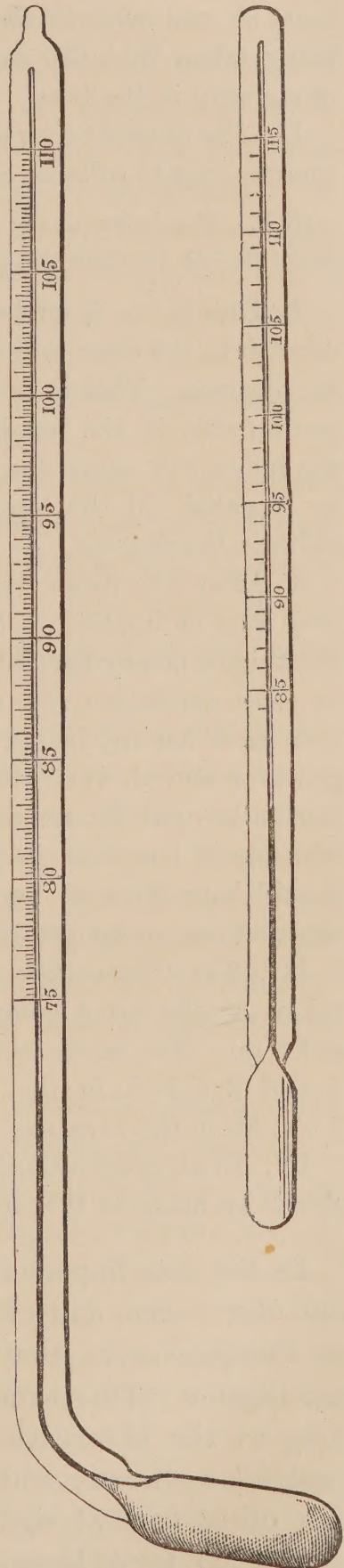


Fig. B.



One half the real size.

than *three minutes*; and the height of the mercury in the graduated stem must be read *while the thermometer is still undisturbed in the axilla*, care being taken that the axis of vision falls perpendicularly on the column of mercury in the tube.

II. *The Straight Thermometer*, Fig. B.—1. Its index must be set *before* commencing to take an observation.

[N.B.—The index is the bit of mercury detached from THE COLUMN IN THE STEM OF THE INSTRUMENT.]

2. This index is to be set by bringing the bit of detached mercury down into the clear part of the stem, just below the lines which indicate the degrees. This is done by taking the bulb and stem of the instrument firmly in the hand, and then by *a single rapid swing of the arm* the index will come down the stem; and this *swing of the arm* must be repeated till the top of the index is at least below the lines which indicate the degrees.

3. After the index has thus been set, the bulb of the instrument may then be applied to the axilla, the surface of the belly, or between the thighs, or any part which is completely covered; and being retained in close apposition (by strapping, if necessary) with the surrounding soft parts for any length of time, the instrument is to be carefully and gently removed, when the *top of the index*—*i. e.*, the end farthest from the bulb—will denote the *maximum temperature* during the period the instrument has been in perfect contact with the parts. The patient should have been at perfect rest in bed for at least one hour before observations on temperature are made.

III. *The Observations* ought to be continuous daily, and regularly taken at the same hour every day, throughout the whole period of sickness. The most useful periods for observation are—(1.) Between 7 and 9 o'clock in the morning; (2.) At noon; (3.) Between 5 and 7 o'clock in the evening; (4.) At midnight.

IV. In all observations of temperature the Pulse and the Respirations should be noted at the same time.

In the less important cases the physician may make at least one observation daily himself, and leave the others to the friends of the patient or the nurse, if either of them are sufficiently intelligent. This arrangement, however, is only justifiable so long as the observations correspond with those typical of the particular disease, and so long as they are in harmony with the other general signs of its course; but as soon as notable deviations from these conditions are observed, the physician ought to make the observations for himself. A difference of

one-tenth R. = 1° Fahr., or of two-tenths R. = 2° Fahr., is not of any practical importance unless it is persistent.

In prolonged and severe cases an examination of the records of temperature made during the course of the disease will recall to mind the nature of the case more effectively than the most detailed written history. For this purpose it is desirable to exhibit on paper the daily thermometric changes, in the form of an angular line or a curve, and to note in the proper places short memoranda of the more important incidents or therapeutic events which have taken place during the progress of the disease. A second and third line may be added, illustrative of the changes in the pulse and the respiration.

In chronic cases, when febrile attacks and their concomitant dangers may be expected, as well as in acute cases, after return of the normal blood-heat *one* daily observation will be found sufficient. This single observation may be best made in the afternoon, or at that hour of the day in which generally some apparent change takes place.

It is advisable to induce nurses, friends, or other attendants on the sick (whenever they seem apt pupils), to make notes of any considerable excitement or restlessness, hot hands, increased heat of head, and to consult at once the thermometer. They may thus, perhaps, tranquillize the patient and his friends when the instrument does not indicate any material increase of heat; but the sudden appearance of any considerable increase of temperature would always be (as we have seen) a fact of vital importance.

It has been recommended by some to place the thermometer under the tongue, as the best place. On the contrary, the cavity of the mouth is the worst place in which the thermometer can be put, because the temperature there is continually varying according to the quantity and temperature of the air used in respiration; and if the atmosphere is cold, and deep inspirations are made, large differences may be observed, compared with the temperature in the axilla. Therefore it is necessary not to trust to observations made with the thermometer in the mouth.

In cases which do not require the most rigorous and extreme accuracy (as cases observed for the sake of scientific information require), *three* to *five* minutes is found quite sufficient in private practice for the application of the thermometer. The simplest

and most convenient way is to heat the instrument before inserting it into the patient's axilla, just as the surgeon heats the catheter before he introduces it into the urethra. This may be done by holding the thermometer in the warm hand, or into water heated to a temperature of nearly 30° R. = 99.5° Fahr.: and, after the instrument is properly placed, be satisfied if *two observations at intervals of one to two minutes give exactly the same result.*

The rapidity with which the mercurial column rises depends on the degree of temperature present. The rapidity of the rise of temperature ought to be noted, as well as the maximum height. If the temperature be above the normal standard, a sensitive thermometer will indicate that fact within the first minute; and as the quickness of the rising depends upon the existing temperature, the physician is able, after some experience in the use of a particular instrument, to form an approximative judgment of the amount of rising of temperature to be expected in any particular case from the slowness or rapidity of the rise of the mercury after half a minute.

II. *Fluctuations of Temperature within the Limits of HEALTH; and the Co-relation of the Animal Heat with the Pulse and the Respiration.*

Several observers in Germany, France, England, and the Tropics, have now determined these fluctuations with great accuracy, so that ample and sufficient data are on record to furnish a standard for comparison in cases of disease.

The temperature of the body is the result of the opposing action of two factors:—1st. Of development of heat from the chemical changes of the food, and by the conversion of mechanical force into heat, or by direct absorption from without; and 2nd. and opposed to this, Of evaporation from the surface of the body, which regulates internal heat (Parkes' *Hygiène*, p. 432).

With reference to the normal range of temperature, our most trustworthy information is mainly due to Valentin and Traube, in Germany; to Edwards, Becquerel, Breschet, and Bernard, in France; and to Dr. John Davy, in England and the Tropics. All agree in stating that the ranges of temperature vary in different parts of the human body; but, as a general practical result, it is equally agreed that in temperate regions the normal temperature *at completely sheltered parts of the*

surface of the human body amounts to 98.4° Fahr., or a few tenths more or less; and a rising above 99.5° , or a depression below 97.3° Fahr., are sure signs of *some kind* of disease, if the *increase or depression is persistent*.

Valentin proved by many experiments that all warm-blooded animals surrounded by an atmosphere of 50° Fahr. to 68° Fahr. have a temperature of about 99.5° in the back of the mouth, the rectum, or other accessible *internal parts*; and at *completely sheltered parts of the surface* it is a degree lower—namely, 98.4° or $.5^{\circ}$. But the most common temperature is about 97.5° , according to Dr. Compton's more recent observations. Dogs have a temperature similar to that of men; so that a knowledge of thermometry in the diseases of animals will prove not less valuable in veterinary pathology than in human, and perhaps more so, inasmuch as such animals are deprived of speech to express their feeling.

The observations of Dr. John Davy, originally communicated to the Royal Society, are the most numerous and extensive in detail on record, which indicate the fluctuations of temperature within the limits of health. An abstract is given in the following table:—

FLUCTUATIONS OF TEMPERATURE WITHIN THE LIMITS
OF HEALTH (DAVY).

I.—IN TEMPERATE REGIONS (ENGLAND).

Period of Day.	Temperature of Body.	Pulse per Minute.	Respiration per Minute.	Temperature of Room.
Morning, 7-8,	98.74° Fahr.	58	16	50.9° Fahr.
Afternoon, 3-4,	98.52° „	55	15	54.7° „
Midnight, 12,	97.92° „	55	15	62° „

II.—IN TROPICAL REGIONS (BARBADOES).

Morning, . 6,	98.07° Fahr.	54	14	76.7° Fahr.
Afternoon, 2,	98.9° „	56	15	83.6° „
Night, 10-11,	99.0° „	60	15	79.8° „

The general result may be stated as follows:—

1. IN TEMPERATE CLIMATES the *Maximum temperature* is in the early morning after waking: it fluctuates till nightfall, and is lowest about midnight; average difference, 0.82° .

2. IN TROPICAL REGIONS the *Minimum temperature* is in the

early morning after waking: it fluctuates, and is highest during the day.

3. *Average temperature* throughout the year, 98.4° Fahr.,—the temperature of the air averaging 55.5° Fahr.

The observations of Dr. Davy and of Edwards have shown that the amount of animal heat may be considerably altered by a number of collateral circumstances. But the great distinction between these alterations of temperature in health, and those which are the result of disease, is, that these variations are generally temporary, and within narrow limits—amounting to mere fractions of a degree—rarely more than from 1.8° Fahr. to 3.6° Fahr. (VALENTIN and DAVY), whereas those which are due to disease are persistent so long as the disease exists.

The following are the collateral circumstances which mainly influence animal heat in our daily life, and which require to be remembered in order that erroneous conclusions may not be drawn:—(1.) *Active exercise* (not carried to the extent of exhausting fatigue) raises the temperature proportionally to the degree of muscular exertion made. (2.) *Exposure to cold* without exercise lowers the temperature. (3.) *Sustained mental exertion* reduces temperature about half a degree. (4.) The amount of heat is also at first reduced after *a full meal* and the use of alcohol; but it rises again as digestion advances. (5.) There are diurnal fluctuations capable of being thus determined. (6.) The temperature of the body rises with the temperature of the air; and sudden transitions from a cold to a hot climate induce a feverish state marked by increase of temperature, accelerated action of the heart, and quickened respiration, especially on bodily exertion. (7.) The average temperature within the *tropics* is nearly 1° Fahr. higher than in temperate regions. (8.) The temperature is more readily and rapidly affected—more sensitive, so to speak—than either the pulse or the respiration; and this is especially the case in disease.

The amount of abnormal increase of temperature is usually proportionate to the degree of frequency of the pulse, and to the other signs of general disease. Yet such congruity of phenomena is sometimes in part or wholly absent or incomplete; and in the cases in which a disproportion or incongruity exists between the increase of temperature and the pulse or other febrile phenomena, it is the accurate measurement of the temperature which is most

of all to be relied upon. As a general rule the co-relation of pulse and temperature may be stated as follows, namely:—"An increase of temperature of ONE DEGREE above 98° Fahr. corresponds with an increase of TEN beats of the pulse per minute, as in the following table:—

Temperature of	98°	. .	Corresponds with a pulse of	60
—	99°	. .	—	70
—	100°	. .	—	80
—	101°	. .	—	90
—	102°	. .	—	100
—	103°	. .	—	110
—	104°	. .	—	120
—	105°	. .	—	130
—	106°	. .	—	140"

III. *Ranges of Temperature in Disease.*

Having satisfied ourselves as to the delicacy and accuracy of the thermometer, and obtained a standard for comparison, we are prepared to appreciate the range of temperature in febrile disease as measured by such an accurate instrument.

The maintenance of a normal temperature, within the limited fluctuations just noticed, under all these varying influences gives a complete assurance of the absence of anything beyond local and unimportant disturbances; and, long before the subject was worked out so thoroughly as it has been, it was often casually observed that any acute disease, however slight, elevates abnormally the temperature or animal heat; "and its undue degree of elevation (as Dr. Davy clearly enunciated) is some criterion of the intensity of the diseased action" (*Physio. Researches*, vol. i., p. 56). In short, it is now placed beyond a doubt by the observations of Gierse, Roger, Valentin, Von Bærensprung, Wunderlich, Friedlander, Virchow, Traube, Jockmann, Greisinger, Billroth, and others, in Germany; by MM. Becquerel, Breschet, and Bernard, in France; by Parkes, Jenner, and Ringer, in this country, that while this preternatural heat varies in amount in different diseases in different persons, and at different times of the same day, it is this preternatural heat which is the essential symptom in fever, which proves fever to be present, and which exists to the extent of 4°, 6°, or even 8° Fahr. over the natural limits of health (which averages 98° Fahr.), and must be judged of by the temperature in the axilla, as indicated by the thermometer. This preternatural heat is never absent in

fever, and without it fever cannot be said to exist. Rigor, which is also sometimes present, is a mere peripheric phenomenon, and the coldness of the skin a subjective sensation, produced by the state of the peripheral nerves, and is not due to any actual decline of temperature. "While the outer parts feel cold to the bystander, the inner parts are abnormally warm. While the outer parts freeze, the inner burn" (VIRCHOW, PARKES, JENNER).

There are many cases now on record in which the physician, without thermometric observation, does not appreciate the existence of fever or of danger. Wunderlich gives numerous examples of this; but long before he brought this subject so forcibly to the notice of medical men, we have the testimony of Dr. John Davy in this country, given quite incidentally, and therefore all the more valuable as an unbiassed testimony of the usefulness of the thermometer in detecting latent disease not otherwise indicated by general symptoms. When Dr. Davy was collecting his extensive observations on the normal temperature of the body, he was surprised to find that one person exhibited for many weeks a persistent temperature of 104° Fahr. This person was a lunatic soldier, and Dr. Davy remembered that the insane do not seem to suffer from cold nor heat like ordinary individuals, and that there are certain organic lesions which are apt to occur in them unaccompanied by the usual symptoms. For example, tubercle and cavities of the lungs occur without cough or difficult breathing; and although no warning nor any indication may be given, the disease runs its course, terminating in death as certainly and as rapidly as if indicated by the ordinary train of symptoms. Discovering then, as it were by accident, that the temperature in this lunatic was as high as 104.5° Fahr., and that his pulse was rapid, Dr. Davy's attention was more particularly aroused; and although the man made no complaint, but had a good appetite, with his digestive functions, so far as was known, acting well, yet disease of the lungs was thus discovered. The lunatic died in a month, of acute tuberculosis, not otherwise expressed by symptoms beyond the great, persistent, and continuous elevation of temperature thus incidentally noticed. There were ulcers of the *larynx* found after death, but there had been no affection of the voice; there were *vomicæ* and *tubercles* in the *lungs*, but there had been no cough; there were *ulcerations* of the *intestines*, but there had been no

diarrhœa; there was disease of the *testes, vesiculæ seminales*, and *prostate*, of a severe kind, but these lesions had been equally latent during life, except hardening and enlargement of the testicle without pain,—all which conditions were only casually observed.

In this very instructive case a temperature of *six degrees* Fahr. above the normal standard was the earliest indication of disease (*Researches, Physiological and Anatomical*, vol i., p. 206).

But it is mainly to Wunderlich, the Professor of Medicine in Leipsic, that we are indebted for an elaborate exposition and persevering advocacy of the usefulness of daily records of the temperature of fever patients, and the constant employment of the thermometer as a means of diagnosis at the bedside. On this subject he has written much, from an extensive experience, embracing at least half-a-million exact thermometric observations, following the continuous progress of individual diseases, the results of which he has compared in more than 5,000 patients. He constantly employs the thermometer in his private practice, and bears unqualified testimony to its sterling value in the early detection of disease, and as often furnishing an important guide to treatment. When the physician once becomes accustomed to the investigation of disease by the thermometer, he regards its daily employment as indispensable, for it imparts a certainty to his observations, attainable by no natural penetration, and which no other method of investigation can convey (*Medical Times and Gazette*, June 19, 1858, and September 28, 1861).

More detailed results are published by the assistants or pupils of Wunderlich, in the *Archives für Physiologische Heilkunde*, 1860, p. 385, and 1861, p. 433; and the principal conclusions have been summed up by Wunderlich himself, in his *Handbuch der Pathologie*. From these sources the information given in this and former editions of this text-book was originally compiled.

Wunderlich gives some striking instances of disease being indicated by thermometric observation before it could be detected by any other means:—

In *ague*, several hours previous to the paroxysm, the temperature of the trunk of the patient's body begins to rise; and when the disease seems to have disappeared, an increase of temperature may be detected periodically, unaccompanied by any other symptom. So long as this periodic rise of temperature continues, the patient is only apparently, but not really cured.

In typhoid fever, during the exacerbations especially, the rise of temperature or its abnormal fall will indicate what is about to happen three or even four days before any change in the pulse, or other sign of mischief, has been observed. A sudden and marked reduction of temperature has thus denoted hæmorrhage from the sloughs of Peyer's patches in typhoid fever, several days before it appeared in the stools. A case of this kind is recorded by Dr. Parkes. It occurred in a female twenty-five years of age. Diarrhœa was considerable, and blood was largely passed in fluid stools the night before the 17th day of the fever. On the morning of that day the temperature was as low as 93° Fahr., rising in the evening to 101° Fahr.

It is rare, however, that a definite diagnosis or prognosis can be based on a single observation; but sometimes it may be sufficient, as in the following instances:—

When the temperature is increased beyond 98.5° it merely shows that the individual is ill, and suffering from some disease, and that when considerably raised, as with a temperature of 101° to 105°, the febrile phenomena are severe; that when a great height is reached, as at temperatures above 105° Fahr., the patient is in imminent danger; and that with a rising temperature above 106° Fahr., to 108° or 109° Fahr., a fatal issue may almost without doubt be expected in a comparatively short time. The highest temperatures before death have been observed in cases of scarlet fever and of tetanus. *acute Rheum 113*

A definitive diagnosis may also be based on a single observation, under the following circumstances:—

Pyæmia A person who yesterday was healthy, but exhibits this morning a temperature above 104° Fahr., is almost certainly the subject of an attack of ephemeral fever or of ague; and should the temperature rise up to or beyond 106.3° Fahr., the case will certainly turn out one of ague, or some other form of malarious fever.

A girl eighteen years of age, supposed to be suffering from hysteria, but simulating a case of cerebro-spinal meningitis. A temperature of 103.5° confirmed the diagnosis of meningitis and negatived that of hysteria. The case terminated fatally (COMPTON). Again, in a patient whose temperature rises during the first day of illness up to 106° Fahr., it is certain he does not suffer from typhus nor typhoid; and of a patient who exhibits the general

typical signs of pneumonia, but whose temperature never reaches 101.7° Fahr., it may be safely concluded that no soft infiltrating exudation is present in the lung.

Again, if a patient suffer from measles, and retains a high temperature after the eruption has faded, it may be concluded that some complicating disturbance is present.

Single observations with other means of diagnosis will often determine whether the disease is one of danger or not.

In typhoid fever a temperature which does not exceed on any evening 103.5° Fahr. indicates a probably mild course of the fever—and especially if the increase of temperature takes place moderately, towards the beginning of the second week. A temperature of 105° Fahr. in the evening, or of 104° Fahr. in the morning, shows that the attack is a severe one, and forebodes danger during the third week; on the other hand, a temperature of 101.7° Fahr. and below, in the morning, indicates a very mild attack, or the commencement of convalescence. In pneumonia a temperature of 104° and upwards indicates a severe attack. In acute rheumatism a temperature of 104° is always an alarming symptom, foreboding danger, or some complication such as pericardial inflammation. In a case of jaundice otherwise mild, an increase of temperature indicates a pernicious turn. In a puerperal female an increase of temperature indicates approaching pelvic inflammation. In tuberculosis an increase of temperature shows that the disease is advancing, or that untoward complications are setting in.

In short, a fever temperature of 104° to 105° Fahr. in any disease indicates that its progress is not checked, and that complications may still occur.

But it is by continuous daily observations that the most important results have been arrived at, especially in the hands of Wunderlich, Greisinger, Traube, Billroth, Parkes, Jones of Augusta, Ringer, and others who are now working most actively in this field of labour.

Certain febrile diseases have been found to have typical ranges or daily fluctuations of temperature throughout their course. In pure unmixed and uncomplicated cases this is found to be so constant that the differential diagnosis may be established by accurate observation of the temperature continuously from day to day. This has now been determined, especially in cases of malarious fever, typhus, typhoid fever, small-pox, scarlatina, measles,

rheumatism, pyæmia, pneumonia, acute tuberculosis. In each of these diseases the temperature is one of the most certain (although not the only) means for determining the real state of the patient as regards morbid disturbances or complications, and a careful observation of temperature from day to day is indispensable for judging as to the prognosis. Frequently it affords the best and ultimate means of deciding in doubtful cases, and often it is the best corrective of a too hasty conclusion: for example, the characteristic variations of the temperature in a typical case of enteric, intestinal, or typhoid fever, are of such a kind that they are not found in any other disease. Intestinal catarrh, severe forms of pneumonia, malarious fever, acute tuberculosis, meningitis, some stages of Bright's disease, may each simulate typhoid fever, and may exhibit some of its most characteristic symptoms; but observation with the thermometer as to the patient's temperature from day to day, will at once, or after a very few days, establish the distinction with certainty.

In the course of many diseases, whose diagnosis has been accurately determined, if the temperature departs from its normal or typical range, the thermometer will furnish the best and the earliest indication of any untoward event, such as the additional development of disease, or other visceral complications in its course.

When once the typical range of temperature (*normal*, as it were, of the particular disease) is determined, a basis is laid for appreciating irregularities or complications in its course in particular cases. For example, a patient exhibits symptoms of fever of the typhoid type, but during the progress of the first week his temperature becomes normal, for however short a space of time;—the occurrence of this event proves that the fever is not what it was supposed to be. Again, a patient may suffer from all the general symptoms of incipient pneumonia; but there still is a doubt as to whether infarction of the lung has taken place. The sputa being suppressed, or not procurable, does not assist the diagnosis. If, however, the temperature is found to be normal, it is certain that no croupous exudation has taken place in the lung, and that there is no pneumonia. Again, if a tuberculous patient has a sudden attack of hæmoptysis, and if the temperature of his body is normal during and subsequent to the attack, no re-active pneumonia, nor any exacerbation of the

tuberculous exudation need be expected. This is a new field open for investigation in cases of phthisis.

Again:—In all cases of convalescence, so long as the defervescence proceeds regularly as measured by the temperature, no relapses need be feared: on the other hand, delayed defervescence in pneumonia, the persistence of a high evening temperature in typhus or typhoid fever, or the exanthemata, and the incomplete attainment of normal temperature in convalescence, are signs of great significance. They indicate incomplete recovery, super-
vention of other diseases, unfavourable changes in the products of disease, or the continuance of other sources of disturbance requiring to be carefully examined into. The onset of even a slight elevation of temperature during convalescence is a warning to exercise careful watching over the patient, and especially for the maintenance of a due control over his diet and actions.

Continuous daily observations by the thermometer thus teach the typical laws of particular forms of fever, and supply the grounds or basis by which it is determined whether any individual case is progressing as it ought to do. Such knowledge can only be acquired by repeated observation of numerous cases; and deviations from the normal temperature in certain diseases are stable in proportion to the typical character and full development of the particular disease. But even in such diseases we may have an increase or decrease of temperature proper to the disease brought about by *accidental* influences. Such instability, however, is only temporary, and of short duration, when the *accidental* influences act but *transitorily*. For example, the temperature proper to the disease may be lowered under the influence of a profound sleep, bleeding, epistaxis, the relief of constipation or of the retention of urine, and the like; or it may be raised after excitement of a mental kind. But any such alterations, unless they are dependent upon a change in the disease-process itself, will become effaced after twelve or twenty-four hours at the most, when the temperature again resumes the typical character diagnostic of the particular disease. In continued fevers the temperature is generally less high in the morning than in the evening.

Stability of temperature from morning to evening is a good sign; on the other hand, if the temperature remains stable from

evening till the morning, it is a sign that the patient is getting or will get worse.

When the temperature begins to fall from the evening to the morning, it is a sure sign of improvement; on the other hand, a rise of temperature from the evening till the morning is a sign of his getting worse.

When it is found, in a bad case of typhoid fever, that some morning about the third week the temperature has fallen to 99.5° , the reparative stage has begun—the healing of Peyer's patches; and when a similar fall of temperature is observed in the evening, convalescence has commenced.

In pneumonia, when a marked fall of temperature occurs in the evening, the period of crisis has arrived.

In measles, when the maximum severity of the eruptive stage has been reached, the temperature falls.

A sinking from a considerable height down to a normal temperature suddenly (within twenty-four hours), occurs in a few exanthemata,—measles, variola, rarely in pneumonia, typhus, and pyæmia.

In tuberculosis, especially in its acute form, the persistent maintenance of an uniformly high temperature will alone show that no arrest in the progress of the disease has occurred.

The co-relation of pulse, respiration, and temperature is of great importance to be determined in many acute diseases; and especially in pneumonia, if the mean of the temperature is not above 104° Fahr., and that of the pulse is not above 120 in a minute, and the mean of the respirations not over 40 in the same time, the case must be considered a slight one; and if the patient is otherwise healthy, he will surely begin to get well in from eight to twelve days, without any medical treatment beyond attention to antiphlogistic regimen.

Convalescence is known to commence when the disease-process ends; and this precise point can only be fixed by continuous thermometric observation. The morbid process does not end till the normal temperature of the body returns, and maintains itself unchanged through all periods of the day and night.

Regularly continuous observations of the temperature exhibit the precise point at which the disease-process terminates, and the degree of its complete development. When this point has been determined on, a retrospective view may be taken of the character

of the disease, as to the purity of its typical form or its complexity, and a prognosis may be hazarded as to the probability or doubtfulness of recovery. The morbid process has not terminated till the normal temperature of the body returns, and remains unchanged in the evenings and throughout all periods of the day. The transition from the febrile state into defervescence is either slow (lysis) or rapid (crisis). A regularly continuous defervescence is always a sure sign of convalescence. Its occurrence will save other investigations: irregular defervescence, on the contrary, indicates a disturbed and protracted course of convalescence, which requires careful watching and judicious nursing.

It is of practical importance to know that the fall of temperature during the period of recovery, in cases of considerable morning remissions, as well as in those of continued defervescence, may be abnormally large, and sink as low as 28° R. = 95° Fahr., or even lower. Such events constitute collapses during defervescence, which must be counteracted by artificial heat, the administration of warm drinks, or even of such stimulants as wine or camphor, unless some unexpected new danger should interfere with an otherwise favourable course of the disease.

During convalescence the recurrence of a high temperature is generally the first sign of an approaching relapse, or the onset of a new disease, the characteristic symptoms of which it may precede by several days. The persistence of even an inconsiderable degree of abnormal temperature after apparent return to health, is a certain, and frequently for a long time the only, sign of incomplete recovery, or the existence of some lingering secondary disease. Thus the temperature should be closely watched during convalescence; and the thermometer should be applied every alternate evening at the very least. As long as the temperature remains normal, nothing need be feared; but every rise of temperature should act as a warning. It may be due to mere error in diet, or to leaving bed too early; but in such cases the temperature soon sinks again, on greater precautions being taken.

Regularly continuous observations on the temperature alone, or in connection with other symptoms, may enable the physician to predict a fatal issue with certainty, or the probably near approach

of death. On this point one of two conditions may be observed,—(1.) The temperature may rise continuously and considerably above 33° R. = 106.2° Fahr., when it is a bad sign; or it may even reach 34° R. = 110° Fahr., when a fatal issue is almost certain; and it not unfrequently happens that, after the apparent occurrence of death, the temperature still continues to rise one or two-tenths R. = 1° or 2° Fahr., the cooling of the body taking place very slowly. Wunderlich records a case of spontaneous or rheumatic tetanus, in which the temperature exceeded the maximum that has ever yet been observed in any disease. The heat only began to increase within the last twenty-four hours before death; but the other symptoms before that time had been very violent, the respirations being accelerated, and the pulse at 102. During the night previous to death the temperature suddenly rose 3.3° Fahr., while the velocity of the pulse and the frequency of the respirations diminished, and the other symptoms did not increase in severity. Shortly before death the heat rose to 110.75° Fahr., the pulse being then at 180; and at the moment of death the thermometer was at 112.5° Fahr. After death the temperature still rose, and was found to be 113.8° Fahr. an hour after the fatal event. It then slowly diminished; and thirteen and a half hours after death the temperature had not yet fallen to the normal average of the living body.

(2.) The temperature may become more or less moderated, while the pulse is increased in frequency, and the other symptoms become more and more threatening. Such diminution of temperature, amidst conditions which do not harmonize with it, must be regarded as a pretty certain sign of approaching dissolution.

But, on the other hand, there are cases in which the observation of the temperature yields the most favourable signs for prognosis. For example, when it is found, in a bad case of *typhoid fever*, that the temperature has fallen some morning to 30° R. = 99.5° Fahr., we know that the reparative stage is entered upon; and when a similar fall of temperature is observed in the evening, convalescence has commenced. In *pneumonia*, when a marked fall of temperature occurs in the evening, it shows that the period of crisis has arrived. When the temperature falls in *measles*, the maximum severity of the eruptive stage has been reached; and when, in the first stage

of *variola*, we observe a quick return to the normal temperature, we may feel certain that a slight form of the disease, free from danger, is likely to ensue.

A decrease of temperature below the normal heat is rare. It happens sometimes transitorily, announcing thereby a favourable crisis, by preceding the return to a normal temperature. It is also met with sometimes during the morning remission of *remittent fever*; also during the apyrexia of *intermittents*; in acute collapse, preceded or not by fever; in chronic wasting diseases; and sometimes, also, on the approach of death, especially in typhus fever.

A remarkable inequality in the distribution of the temperature over different parts of the body (face, hands, feet, &c.) may occur during the shivering preceding fever, in collapse, and in the agony of approaching dissolution. Sometimes, also, such unequal distribution may occur in disorders of the chest and abdomen, in some local skin diseases, and in partial paralysis. This fact is not of importance or utility for diagnosis or prognosis; but it requires to be known, in order that erroneous conclusions may not be drawn.

IV. *Of the Ranges of Temperature in Diseases where Fever is present, as related to the Amount of the Excreta.*

The particular degree of heat and the waste in every febrile disease are represented by—*something*. The physician sees the fevered patient wasting before his eyes. Every tissue is wasting, and, in co-relation with the excessive generation of heat, how is this waste expressed? As a rule, it is expressed by the amount of *excreta*.

To Dr. Parkes in this country, to Dr. Jones of Augusta (in cases of malarious fever), to Virchow and Wunderlich in Germany, is the merit mainly due of having demonstrated, by clinical and experimental observation, that “the morbid development of heat, *as measured by a thermometer*, is associated in some cases with MORE abundant, in other instances with LESS abundant *excreta* from the body than in health;—that the temperature and the amount of the excretions bear some undetermined relation to each other;—and that the loss of weight of the patient is due to increased and rapid elimination of material with increased tissue-change, associated with the increase of temperature.

So far as physiological facts have elucidated the normal generation of heat in the healthy body, so far has the abnormal generation of heat essential to the *febrile* state been clearly made out. In health the normal temperature produced by chemical change in the body is represented in the excretions by so much *urea, sulphuric acid, carbonic acid, excretive volatile acids* of the skin, and the like; but in the febrile body a higher temperature is represented in the excretions, in some cases by a larger, and in others by a smaller quantity of *urea, sulphuric acid, and probably carbonic acid* * (PARKES).

The most opposite statements have thus been made regarding the amounts of the excretions in fever, compared with the quantity excreted in health; and at present many excellent observers hold that these excretions are always, and of necessity, increased; others, no less exact, affirm that they are invariably, or almost always, diminished. Such discrepancy of statement is due, in the first instance, to the difficulty of collecting and measuring exactly the amount of all the excretions. "Two of the excretions, the cutaneous and the pulmonary, cannot be collected and measured with anything like the accuracy necessary in such an inquiry: even in health such an inquiry is difficult, and in fever it is almost impossible." By careful and accurate observation at the bedside, however, Dr. Parkes has been able to obtain very close approximative data to found his conclusions upon relative to the increase or diminution of the excretions. He assumes that when the respirations are not

* In the study of special diseases the student ought frequently to estimate the quantity of *excreta* by the urine, as one of the best methods for enabling him to appreciate the changes which go on in the body during disease. To aid him in prosecuting such researches, he is recommended to consult the work of Dr. Parkes *On the Composition of the Urine*, and to follow the directions given on the *Examination of the Urine*, towards the end of the second volume of this text-book, for obtaining quantitative results by the volumetric method.

Average quantity of urine passed in twenty-four hours,				52 $\frac{3}{4}$ to 56 ounces.
Average amounts of solids " " "				945 grains.
"	"	<i>Urea</i>	" " "	512 "
"	"	<i>Chlorine</i>	" " "	126·76 "
"	"	<i>Free Acid</i>	" " "	33 "
"	"	<i>Phosphoric Acid</i>	" " "	48·80 "
"	"	<i>Sulphuric Acid</i>	" " "	31·11 "
"	"	<i>Uric Acid</i>	" " "	8·5 "
Specific gravity, ...				10·20.

quickened (*i. e.*, about eighteen times in a minute, or about one act of respiration for every four beats of the pulse), and when the skin is not evidently sweating, the excretions by these two organs are not increased; and, on the other hand, an increased excretion by these organs may reasonably be inferred if the exercise of their function is unusually active, and if there are tolerably copious perspirations. The other two excretions, namely, the urine and intestinal discharges, can be measured with accuracy, and the urine in particular is a valuable index of the metamorphoses of tissue. The *urea* alone represents two-thirds of the whole quantity of *nitrogen* which passes off; the *sulphuric* acid (the sulphates of the food being accounted for) represents almost entirely the oxidation of sulphur; and the oxidized phosphorus of the body passes out in great measure, though not altogether, as urinary phosphoric acid. Therefore a careful examination of the urine, and of the intestinal discharges, with an approximative estimate of the pulmonary and cutaneous excretions, give sufficiently extensive and accurate materials for the question at issue.

The products excreted are thus of such a kind as to be eliminated, some by *the lungs*, some by *the skin*, some by *the bowels*, and some by *the kidneys*, and rarely by two or more modes of excretion—for when the discharges from the *skin* or *bowels* are profuse, those by the *kidneys* are deficient, as in the last two cases recorded in the following Table I., in which the augmented excretions are printed in italics. The facts thus so carefully observed by Dr. Parkes, confirmed by Alfred Vogel, Heller, and others (but chiefly in regard to the excretion of urea only), justify the conclusion—“*That increase of temperature may be attended with increased elimination; and therefore presumably with increased tissue-change.*”

TABLE I.—ABSTRACT OF CASES OBSERVED BY DR. PARKES IN WHICH SOME OF THE EXCRETIONS ARE INCREASED IN CONSEQUENCE OF THE FEBRILE STATE.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatism.	Fahr. 3°	Not noted.	<i>Sweating profusely.</i>	Discharge as usual.	Solid matter excreted greater than in health by 100 grains, and due to urea and sulphuric acid.
Rheumatism.	Fahr. 3°	Not noted.	<i>Sweating profusely.</i>	Discharge not diminished.	Solid matters excreted greater than in health by 200 grains, and due to urea and sulphuric acid.
Typhoid Fever.	Several degrees.	<i>Rapid.</i>	<i>Moist.</i>	Not increased.	Increased by 60 grains.
Erysipelas of Head and Face.		<i>Quick.</i>	<i>Moist.</i>	Unaltered.	Considerably augmented.
Febricula.	Fahr. 3°	Normal.	<i>Enormously augmented.</i>	Confined.	Solids less than normal by 91 grains.
Typhoid.	Fahr. 3°	Not noted.	<i>Sweating and Sudamina.</i>	<i>Diarrhœa profuse.</i>	Solids less by 73 grains.

TABLE II.—CASES OBSERVED BY DR. PARKES IN WHICH THERE WAS DIMINUTION OF THE EXCRETIONS.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Bronchitis of both lungs.	Fahr. 2·6°	20 Respirations per minute.	Not increased.	Not increased.	Less by 112 grains.
Pneumonia acute sthenic.	Fahr. 5°	30 per minute; expectoration scanty.	Slightly moist.	Confined.	Less by 220 grains.
Typhoid Fever.	Considerable.		No sweating.	No diarrhœa.	Below normal amount.
Acute Rheumatism.	Fahr. 4°	Tranquil breathing.	Inconsiderable.	Bowels quiet.	Very small amount of urinary solids.

The abstract given in Table II. shows that another conclusion is equally legitimate, namely—"That the products of metamorphoses, as judged of by the excreta, may be diminished in febrile cases; and these apparently discordant statements are capable of being explained in various ways. In the first place, it is evident that more chemical change may go on in the body than is represented by the excreta. The metamorphosis of blood or of tissues may not be carried to the point of forming those principles which can alone pass through the eliminating organs. A vast amount of imperfectly organized compounds may be formed and retained in the system, circulating with the blood or thrown upon certain organs." Thus there may be increased metamorphosis with lessened elimination. Several pathological facts point to such a conclusion.

1. It is in such febrile cases, with diminished excreta, that, at a later period of the disease, copious discharges from one or other of the eliminating organs occur. Thus, in the case of pneumonia referred to in Table II., severe spontaneous diarrhoea came on; and many other cases are quoted, with similar diminution of the excretions at the period of increased febrile heat, in which violent purging, sweating, or diuresis, with increase of urea and of sulphuric acid, subsequently occurred. Such discharges occurring during the progress and towards the termination of a febrile disease have been termed *critical*, the occurrence being called a *crisis*; and the particular day on which it happens, counting from the day of seizure, has been called a *critical day*. The term *crisis* or *critical* is applied because the occurrence of such discharges is usually coincident with more or less sudden fall of temperature, and general improvement in the condition of the patient, whose convalescence dates from the critical day; when, in common language, his disease is said to have "*got the turn*." In such cases, therefore, a large amount of partially metamorphosed substances are retained until they are suddenly discharged, and the system freed from the noxious compounds. Coincident with the critical discharge, the temperature is found to fall.

2. But in another class of febrile cases retention of the products of metamorphosis is not followed by such a fortunate *critical* issue. At a later period in the history of some febrile cases, with diminished excreta, it is not uncommon for *secondary inflammatory* affections to occur, as if the blood were more con-

taminated; and it is sometimes observed that in a patient whose excreting organs are acting copiously, there occurs a diminution of excretion when a simultaneous or subsequent development of local disease becomes manifest.

The following Table of Cases recorded by Dr. Parkes is interesting from the exactness of the observations and the coincidence of the local lesions with suppression and retention of excreta during febrile states:—

TABLE III.—TABLE OF CASES OBSERVED BY DR. PARKES TO SHOW LOCAL LESIONS COINCIDENT WITH SUDDEN RETENTION OF THE EXCRETIONS IN FEVER.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatic Fever. Observed on the 5th, 6th, and 7th day of the disease.	Fahr. 2° Fahr. 2°	No record.	Sweating profuse. Lessened on the 8th day.	No intestinal discharge.	While 400 grains more than in health were being daily excreted, <i>suddenly</i> on the 8th day a diminution of the solids took place by 602 grains; and coincident with this diminution a local lesion became developed (<i>angina faucium</i>). Next day the excretion augmented, and the local affection subsided.
Typhoid Fever.	Fahr. 5° Fahr. 5°	No record.	Great sweating. Lessened much.	Diarrhoea profuse. Diarrhoea ceased.	While the average daily excretion for 8 days was 422·348 grains, a gradual diminution continued for three days, to the daily extent of 78 grains, when pleurisy came on.
Rheumatic Fever.		No record.	Sweating moderately.	Unchanged.	Considerable quantity of urine passed, containing an <i>excess</i> of solids: a sudden and great diminution both of the solids and fluids of the urine took place, when the joints again began to suffer, and pleurodynia supervened.

Thus it is evident, from these carefully recorded observations,

“that diminished excreta in fever are to be referred to retention of such excreta, and not to a want of formation; and that while the amount of excreta (capable of being measured) may in fact be small, the amount of tissue-change may nevertheless be great.”

The practical lesson, so often insisted on by the older physicians,—*“never to lock up the excretions,”*—is thus demonstrated with scientific exactness. Another general and practical conclusion is, that the *febrile heat* cannot be measured by the amount of the excretions as a whole, nor by any ingredient of them in particular; but must be estimated in co-relation with age and body-weight. Under the same degree of heat and in the same disease different patients pass very different quantities of *urea*, *uric acid*, *sulphuric acid*, *phosphoric acid*, *cutaneous* and *intestinal excretions*. The same observations may be made regarding men in health. No two persons pass exactly the same amount of excretory products.

The nature of these excretory products of the febrile state teach us, however, that it is the albuminous or nitrogenous tissues of the body which are being destroyed; for the excretory products of the urine are the representatives of the azotized structures. The amount of these excretory ingredients varies considerably from day to day in fever, exactly as in health. Often there is a regular gradation of increase and decrease: the *urea*, for instance, may, for two or three days, slightly but regularly diminish in amount, and then suddenly augment to its highest point, again slowly to fall. The same fact may be observed with the *sulphuric acid*; and Dr. Parkes is led to believe that, both in health and in disease, a certain periodicity, having a range of three or five days, is connected with these gradations of increase and diminution.

The largest amount of *urea* excreted in twenty-four hours in the febrile state is recorded of a case of *pyæmia*, by Alfred Vogel, namely, 1,235 grains. The largest amount observed by Dr. Parkes was in a case of typhoid fever, in which it amounted to 885 grains. The largest amount of *sulphuric acid* recorded by the same observer, when no medicine was taken, was in a case of rheumatic fever. It amounted to 52.668 grains; and under the influence of liquor potassæ in the same disease, he has known this excretion rise to 70 grains—more than twice as much as in health. The largest amount of *uric acid* excreted during a

febrile disease in twenty-four hours, as recorded by Drs. Parkes and Garrod, has been 17·28 grains.

“The amount of tissue destroyed in order to furnish such quantities of excreta must be enormous; and if it is recollected that little or no food is taken by the *feverish* patient, and, therefore, that no materials are supplied for the reconstruction of the textures thus melting away three times more quickly than in health, the rapid loss in weight in fever, and the impaired nutritive condition of every organ at its close, will be at once evident.”

It is not yet determined where the increased destruction of the albuminous textures takes place; that is, whether it occurs in the blood or in the organs themselves. It is only known that both the albumen and the red corpuscles of the blood are lessened in amount at the end of a febrile disease; and of the various tissues none appear to waste so fast as the muscles, and especially the involuntary ones (*e. g.*, the heart in typhus fever). The fat of the body is rapidly absorbed in fevers; and Virchow asserts that the bones also become lighter. While it is known that much of the metamorphosis of these tissues takes place in the normal way, it is also probable that there is an unhealthy or perverted metamorphosis which leads to the appearance of compounds in the excretions, either altogether foreign to the body or foreign in respect of place and time. There is evidence of this in the peculiar smell of the perspiration, in the peculiar colouring matter of the urine, as well as in the occasional excretion by it of hippuric as well as of lactic, valerianic, and other organic acids.

Next to the occurrence of preternatural heat in fever, the *excessive retention of water in the febrile system* is perhaps the most remarkable and constant phenomenon. Notwithstanding the large amount of water frequently taken to quench the extreme thirst, the quantity of the urine is lessened, and is even scantiest when the skin is driest; and the “concentration of the urine appears to be almost as good an index of the amount of fever as the temperature itself.” The excretion of water by the skin is, as a rule, diminished; and it is a well-known clinical fact that the skin is drier than usual in febrile affections. Very early in the febrile state the buccal mucous membrane also becomes sticky, and the amount of saliva diminishes; and the decrease in the quantity of the gastric fluid during fever has been proved by the

well-known experiments of Beaumont on Alexis St. Martin. The intestinal juices, like the gastric, are also probably diminished, for the stimulus of food is taken away, constipation prevails, and the fæces are dry (PARKES).

This retention of water in the system cannot at present be explained; but Dr. Parkes has suggested that it may possibly be due to the presence in the blood (or tissues generally) of some intermediate waste product of the febrile body, of some substance which (like gelatine) has a powerful attraction for water.

Besides water, there is reason to believe that chloride of sodium is retained to a certain extent in fever, or that it passes off less readily with the urine; but much has yet to be learned of the nature of fever from investigations regarding the chemistry of the *excretions*, of the *secretions*, of the *blood*, and of the *organs*.

The Urine in Fever.—The general characters of this excretion proper to the febrile state are deficiency of water, increase of solids, if they are not retained, and especially of the *urea*, the *uric*, the *sulphuric*, the *phosphoric*, and the *hippuric acids*. The *pigments* also are increased; and the *chloride of sodium* is diminished. The deep colour of febrile urine has usually been attributed to its concentration; but if febrile urine be diluted to the usual amount of fluid contained in healthy urine, it is still darker than normal urine. The colouring matter has been shown by Vogel to be increased sometimes fourfold, and it appears to contain more carbon than usual. This colouring matter in febrile urine is peculiar, and does not give any of the re-actions of the bile-pigment. It may, according to Dr. Parkes, be considered as a measure of the metamorphosis of the blood-globules, which in some cases may thus be four times as rapid as in health.

Another important fact connected with the chemistry of the urine in fever is *the augmentation of its free acidity*, as measured by its neutralization with soda.

The Blood in Fever.—The most trustworthy and interesting facts connected with the chemistry of the blood in fever are,—(1.) A diminution of the alkaline salts, as shown by Becquerel and Rodier in inflammation; (2.) A diminution of alkalinity of the serum, as shown by Cohen; (3.) The diminution of the albumen after the fever has lasted for some time, with a commensurate increase in the water of the serum; (4.) A diminution in the numbers of the red corpuscles of the blood; (5.) In certain specific

fevers the presence of *uric acid* has been detected; for instance, in rheumatism, by Dr. Garrod.

Of the **Pulmonary Excretion**, in the febrile state, little is known. Some have found the carbonic acid augmented, others have found it diminished. Dr. Wilks, of Guy's Hospital, found that the ratio of respirations to the pulse is always increased, and that the pulse may be descending while the respiration remains high. Such phenomena he considers indicative of a positive increase of function of the lungs.

It is important to determine when the blood becomes affected in fever. It has been, and still is, a favourite opinion to refer the *origin* of fever to primary disease of the blood; and in almost all specific diseases, such as in the *miasmatici*, a fever-making cause appears to enter the blood; at least, writes Dr. Parkes, it may be proved to enter in several cases; and a strong analogical argument can be proved of its entrance into others. The fever-making cause also reproduces itself in the blood, or in some organs; and it is now generally admitted that the first action of the febrile cause is on the blood.

The **Nervous System** seems to play so important a part in fever that Virchow, in his definition, states that the essential phenomena *must have* their immediate cause in changes of the nervous system.

It is very difficult to substantiate this position, but the following general results prove the great influence of the nerves in febrile affections. Taken individually, they, no doubt, will impress different minds with different degrees of force, while collectively they cannot fail to furnish an argument in favour of the essential participation of the nervous system in fever:—

1. There is the generally received physiological law, that nerves regulate the metamorphosis of tissue and the production of heat, both of which are altered in fevers (HELMHOLTZ, LUDWIG, BERNARD).

2. There are those experiments on the vagus nerve which bring about febrile phenomena, such as increased cardiac action, pulmonary congestion, anorexia, and nausea (BERNARD, PAVY).

3. There are those arguments derived from the various symptoms which announce, accompany, or terminate fever. (a.) The remarkable depression, apathy, sense of exhaustion and debility which usher in the febrile state. (b.) The shiverings, the

contraction of the superficial vessels and of the skin. (c.) The increased rapidity of the heart's action, and the relaxation of the vessels, which soon follows the stage of contraction just noticed, or occurs without it. (d.) The congestion of the lungs. (e.) The periodicity of some of the phenomena of fever, and the occurrence of death or recovery on so-called critical days. (f.) The abnormal state of the secretions.

4. The fearfully rapid death which sometimes ensues in the early stage, from some unknown cause, may with justice be referred to profound nervous lesions; for there is great prostration, a galloping and early-failing pulse, and an excessively rapid respiration.

5. The effect of certain remedies, such as quinine, upon periodical febrile phenomena.

Conditions which combine to produce the Complex Phenomena of Fever.—1. The entrance into the blood of a morbid agent, and the alteration of the blood to a certain extent under its influence, come first in the order of events. Perhaps this occurs under the incubative period, when often there is no rise of temperature, no fever; that is, when no appreciable alteration of the general health can be discovered. The nature of the change in the blood is unknown.

2. When the change in the blood has reached a certain point, the nervous system, or rather that part especially connected with nutrition and organic contractility, begins to suffer changes in composition, which probably paralyze, impede, or destroy the normal molecular currents. When this occurs, the nervous symptoms of weakness, depression, rigors, contraction of some parts and vessels speedily followed by relaxation, mark the stage of invasion.

3. Various parts simultaneously, especially the muscles, and probably some of the organs, deprived in greater or less degree of nervous influence, begin rapidly to disintegrate, and by their disintegration preternatural heat is produced. Little or no fresh material is assimilated to compensate for the loss; great muscular prostration ensues; and destruction of tissue is increased by the accelerated action of the heart.

4. This metamorphosis is aided, in most cases, by the condition of the vagus and vasi motor nerves, which cause increased action of the heart and dilatation of the vessels.

5. The contamination of the blood, already produced by the morbid agent, is increased by the check which the normal extra-vascular currents experience, by the pouring into the blood of the rapidly disintegrating tissues, and by the continued action of the morbid agent, which in almost all cases appears to act more rapidly and more powerfully in blood rendered impure in any way, either (as shown by Dr. Carpenter) by retention of excretions, absorption of septic substances, or, as in fever, by the too rapid metamorphosis of tissue.

6. The various organs suffer (apart altogether from specific changes), and must produce increased deterioration of the blood. Thus the lungs are congested in so many cases that we can scarcely suppose proper aëration to go on; the liver would seem, from Frerich's observations, to be, in some cases at any rate, in a most abnormal condition, and to produce compounds, such as leucin, unknown in health; and the spleen in many fevers, if not in all, enlarges (in persons of a certain age), and is congested, possibly even to extravasation.

7. Food being almost withdrawn, the various alkaline and neutral salts no longer pass into the system.

8. Non-elimination of the products of tissue-metamorphoses may give rise to cerebral symptoms and local inflammation.

9. On the elimination of the fever poison and of the products of the tissue-metamorphoses, the nerves resume their normal functions, the undue consumption of tissue is checked, and the patient regains strength and weight.

10. When coma, delirium, or stupor present themselves in the course of fever, it is the custom to refer these symptoms to the action of the fever poison on the brain; but the cerebral functions are more probably deranged not alone by the fever poison, which was the first and the necessary link in the pathological chain, but by the accumulation in the blood of the products of tissue-metamorphosis, and by the perverted and defective nutrition of the brain itself. Hence the symptoms in the advanced stage of many fevers are closely assimilated, although the primary poisons have been perfectly distinct (PARKES, MURCHISON).

All these events tend to render the febrile state an extremely complex one, and its investigation difficult. Our science is often spoken of as an exception to the exactness or comparative

certainty of the (so-called) physical sciences; but nothing can demonstrate more clearly the claims of the Science of Medicine to exactitude and certainty than the advances made in recent years in our knowledge of the natural history of febrile diseases—their causes, their modes of propagation—their development, natural course, and termination.

Improved methods and instruments of research, and more extensive clinical instruction at our schools of medicine, have contributed to this end; and perhaps nothing demonstrates this exactness and certainty better than the advances made in our knowledge of the "thermometry of disease," and the co-relation of temperature with other morbid phenomena. By numerous careful observations it is now clearly established that the determination of the co-relation of the pulse, the respiration, and the temperature of the body in disease is of the greatest practical importance, and especially when regarded in relation to the *excreta*. By such observation the natural course and termination of many diseases can be predicated with great certainty; and so our knowledge becomes more exact as to the nature and treatment of diseases. The pulse, the respirations, and the temperature, all and each of them, represent forces at work in the living body, all of which are capable of being measured with great exactness; and such measurements show how closely such expenditure of forces is related to the *excreta*, which represent the waste of our tissues in health and disease. The student or physician who continues to disregard the aid of thermometry in the diagnosis of febrile disease, or the military medical officer who ignores its value in the appreciation of feigned diseases, such as rheumatism, may be compared to the blind man guiding himself. By means of great practice and intelligence, the blind man will often proceed rightly; but the advantages of being able to see clearly are proverbially above all price. The necessity of using the thermometer will also soon become known to the general public, and patients will become dissatisfied if all known means of investigation are not employed in appreciating the nature of their malady. For many years the German student and physician has been familiar with its use; but, with the exception of Dr. Parkes and the pupils he taught when clinical professor in University College Hospital, the usefulness of the thermometer in recognizing febrile diseases does not seem to have

been hitherto sufficiently appreciated in the medical schools of this country.

SECTION II.—INFLAMMATION.

Definition.—*A complex morbid process characterized,—(1.) By a suspension of the concurrent exercise of function among the minute elements of the tissue involved; (2.) By stagnation of the blood and abnormal adhesiveness of the blood-discs in the capillary vessels contiguous to the tissue-elements whose functions are suspended; (3.) By contraction of the minute arteries leading to the capillaries of the affected part, with subsequent dilatation and paralysis of the contractile tissue of the affected blood-vessels. The nutritive changes between the blood and the minute component elements of the affected tissue become visibly altered, and although an appreciable interstitial exudation does not necessarily follow, yet a constant tendency betrays itself to the occurrence of an interstitial exudation, but which, under proper regimen and proper remedies, is often abortive. When an exudation follows as a result of the inflammatory state, it is apt to be associated with an unhealthy condition of the blood, and of the blood-plasma, and to be associated with varied forms of new growth, according to,—(1.) The elementary structure in which it occurs; (2.) The special zymotic, constitutional, or local disease with which this complex morbid process may co-exist; and (3.) According to the progress of the inflammation, the amount and suddenness of the effusion, the extent of tissue involved, the diminished vascularity, and the powers of absorption of the surrounding parts.*

Pathology.—As it is not possible clearly to define the limits of natural processes, it is not possible to give a correct definition of inflammation. It is a process the most important of all morbid states; and a knowledge of its phenomena, the laws which regulate its course, and the relations which its several events bear to each other, have been always considered as “the keystone to medical and surgical science,” and the “pivot upon which the medical philosophy of the time has revolved.”

It is not wonderful, therefore, that much has been written on this subject, more especially since microscopic research has been brought to aid in the investigation. Among the many who have investigated this morbid process with success, and by whose original observations its study may be said to have begun, the

names of Wilson Philips, John Thomson, Gendrin, Kaltenbrunner, Gerber, and Müller; and more recently those of Alison, Lebert, Gulliver, Addison, C. J. B. Williams, Bennett, Wharton Jones, Henle, Virchow, Paget, John Simon, and Joseph Lister, are well known; and no account of inflammation can be complete which does not embrace the results of the labours of these men.

The early experiments which illustrate the nature and phenomena of inflammation have been made chiefly on the web of the frog's foot, as well as on the folds of the frog's mesentery; and the phenomena are found to correspond in all essential points with the results of experiments performed on the more or less transparent parts of warm-blooded animals, such, for example, as the wings and ears of bats, the ears of rabbits, the mesenteries of these animals, and the brains of rabbits and of pigeons. As a general result of such experiments and observations, it may be stated that the chief constituents of the inflammation-process are to be found in altered conditions of the healthy nutritive changes—the phenomena of the abnormal state becoming more or less obvious by the redness, swelling, heat, pain, impairment of function on a large scale, and sometimes exudation in the part affected.

Phenomena and Theory of the Inflammatory Process.—The process is one in which many stages of morbid action are passed through, and which reaches its acmé when the serum of the blood and the *liquor sanguinis* transude through the walls of the blood-vessels of the inflamed part, without rupture, into the surrounding texture. This has been termed “exudation.”

The series of complex changes through which the inflammatory process is seen to proceed, as observed in the transparent parts of animals under the microscope, are found to occur nearly in the following order:—1st. The beautiful experiments and observations of Mr. Joseph Lister, Professor of Surgery in the University of Glasgow, clearly prove that a suspension of the concurrent exercise of function among the minute elements of the tissue involved is the primary lesion in the congestion of inflammation, and which immediately leads to—2d. Inflammatory derangement of the blood, which, in the vicinity of the impaired tissue-elements, tends to assume the same characters as blood always assumes when it is in contact with ordinary solid matter, and

which renders it unfit for transmission through the blood-vessels. But a return of the tissue-elements to their usually active state will be associated with a restoration of the blood to the healthy characters which adapt it for circulation (*Royal Society, June 18, 1857*). 3d. The arteries of the affected part are narrowed, and the blood flows through them with greater rapidity. 4th. The same vessels subsequently become enlarged, and the current of blood is slower, although uniform. 5th. The flow of blood becomes irregular. 6th. All motion of the blood ultimately ceases, and complete stagnation ensues. 7th, and lastly, The liquor sanguinis may be exuded through the walls of the blood-vessels, sometimes accompanied by the extravasation of blood-corpuscles, owing to rupture of the capillaries.

These different phenomena are associated with the production of the more obvious symptoms, namely, redness, pain, heat, and swelling. But although these changes are here mentioned consecutively, it is not to be understood that in every instance of inflammation such changes can be traced in distinct succession. The changes are to be studied as *nearly concurrent*, rather than as a distinct *series of events*, of which each stands in the relation of a consequent to one or more of its antecedents; so that, starting from impaired function of the elements of tissue, to stagnation of blood in the capillary vessels, we must observe the various stages in the process almost as concurrent phenomena, which, for the purposes of study, are here enumerated in sequence.

An analysis of these concurrent phenomena has shown that the conditions for the healthy nutrition of the part are materially changed, being somewhat as follows:—

I. The supply of blood to the part is altered,—(1.) By the changes in the blood-vessels; especially the narrowing of the arteries and subsequent enlargement of the capillaries; (2.) By the mode in which the blood moves through them.

The narrowing of the arteries, in the first instance, may be demonstrated under the microscope, by the application of warm water simply to the web of the frog's foot; and the same phenomena are presumed to occur in man, for the following reasons:—Sudden operations of the mind and the application of cold produce paleness of the skin—an effect which can only arise from contraction of the minute arteries, and the diminution of the quantity

of blood thereby conveyed by them. The subsequent enlargement of the capillaries is presumed to be a constant event in the inflammation of a part. It usually extends to some distance around what may be considered as the chief seat, centre, or focus of diseased action, and in some textures the enlargement and reddening are confined to the vascular parts in the vicinity. To this condition of the blood and blood-vessels is to be ascribed the usually first observable symptom of inflammation in a part, namely, the redness. But there are also many circumstances under which inflammation has existed, and yet no redness is apparent in the part itself. Thus we often open the body of a patient that has died of phthisis, and find the intestine ulcerated; but, so far from being redder, it is paler than natural, and, so far from being thickened, it is thinner than usual. We often find the cartilages of the joints ulcerated, and yet not a trace of a red vessel. In cases of bronchitis, with purulent expectoration, if the lungs be washed so as to remove the morbid product, the most experienced anatomist may be unable to determine whether the parts are in a state of health or disease. Take the arterial system, and how often do we find the aorta thickened and thinned, softened and indurated, ulcerated, and its elasticity entirely destroyed, and yet not a red vessel to be seen; and when the patient has neither complained of the slightest sensation of pain, nor of any feeling of heat in the part during life? A large abscess may form in the brain or areolar tissue, or pus may be effused into the cavity of the abdomen, without any appearance of redness, or even evidence of having been preceded by any suffering. Although in certain parts, as the cornea and the articular cartilages, the ulcerated intestine or the bronchi, the arterial tissues, and the seats of abscesses, the previous existence of inflammatory action is obvious from the effects produced, and where no blood-vessels existed obvious to the eye, assisted or not by the microscope, yet it is, for the most part, found that enlargement of the blood-vessels of the adjacent parts, and especially of those from which the diseased part derives its nutrient supply, is a constant phenomenon, purely functional, and which appears to be developed indirectly through the medium of the nervous system. In inflammation of the cornea, for instance, the blood-vessels of the sclerotic and conjunctivæ are enlarged. In ulceration of the articular cartilages, the surrounding synovial

membrane and the articular extremities of the bones are more fully pervaded with enlarged blood-vessels. The *vasa vasorum* of the aorta round the morbidly thickened part are also the subject of enlargement, and the channel of increased supply of blood. There is, therefore, no doubt that the conditions favourable to the existence of redness are always present to a greater or less degree at the early period of inflammation; and whether the redness be always present or only slightly perceptible, the same impairment of function among the minute elements of the tissue, and increased adhesiveness of the blood-discs, take part in the development of the inflammatory process.

The enlargement of the blood-vessels varies. It may be hardly perceptible, or it may increase their diameter to two or three times their natural size. John Hunter established this stage of the inflammatory process in the ear of a rabbit, by thawing it after it had been frozen: the rabbit was killed during the process, and the head being injected, the two ears were removed and dried. Wood-cuts representing the comparative conditions of the two ears may be seen in the first volume of Paget's *Surgical Pathology*, page 295. The blood-vessels of the inflamed ear became greatly larger than those of the healthy one, and it was found that arteries before invisible, in the healthy state of the rabbit's ear, were brought clearly into view during the stage of the inflammatory process.

The redness of an inflamed part is of various intensity and shade, according to the degree of the inflammation, its stage, and the structure of the part affected. Its shades pass from a light rose-colour to a deep crimson, or even purple. It assumes the form of points where congeries of minute blood-vessels are concerned; or streaks, as where the vessels of fibrous structures are inflamed, as in tendon; or a series of minute and fine ramifications, as in synovial structures; and generally it may be stated that the form of the redness derives its character from the normal arrangement of the capillaries of the part. The redness is most intense towards the centre of diseased action, gradually softening down towards the circumference, where the conditions of health exist. This gradual shading off serves to distinguish the redness of inflammation from the redness of extravasation. The margin of an extravasation is defined, its redness cannot be removed by pressure; while the disappearance of inflammatory redness under

pressure is, to a certain extent, a measure of the activity of the circulation in the part. The brighter hues generally attend ordinary active inflammation;—the darker hues of inflammatory action are generally associated with some specific cause of disease, a feeble action of the parts, or a tendency to gangrene. The increased depth of colour is mainly due, in the first instance, to the congestion and stagnation of blood in the existing vessels, and not in any measure to the formation of new ones. The redness, however, always appears more than proportionate to the enlargement of the blood-vessels; and we find that the red corpuscles are intensely adherent in the enlarged capillaries.

The dilated vessels of an inflamed part appear crammed with red corpuscles, which lie or move as if no fluid intervened between them, or as if they were imbedded in a hyaline substance due to the solidification of the fibrine of the liquor sanguinis. An increase of redness is sometimes seen to depend upon extravasation of blood, or the effusion of the colouring matter of the blood-corpuscles, as well into the spaces between the blood-corpuscles as into the adjacent tissue through the walls of the blood-vessels. Lastly, the redness is sometimes intensified (as HUNTER first suggested, and microscopic examination subsequently proved) by the passage of the blood, unchanged, from the arteries into the veins. No new formation of blood-vessels is necessarily concerned in the redness of inflamed parts. It is only when inflammation has subsided that new vessels are formed, and pass into any new growth of tissue which may have arisen, as if for its nutrition, development, and continued growth, or to effect its subsequent removal, degeneration, decay, or absorption.

Peculiar changes of shape are associated with enlargement of the blood-vessels, consisting chiefly of tortuosity of distribution and aneurismal or varicose dilatation. The aneurismal or varicose state is seen to take place most frequently in the soft textures, as in the brain, where it is a frequent condition of the inflammatory red softening (KÖLLIKER and HASSE); and in subcutaneous tissue, the points of what appears to be extravasated blood are aneurismal dilatations of capillary vessels filled with the red corpuscles (LEBERT).

These varied conditions of the blood-vessels affect the motion of the fluid in the part, and consequently the supply of blood for the purposes of nutrition. Generally it may be stated that there is

stagnation of the blood in the focus or centre of severe inflammation ; and this stagnation is surrounded by a state of fulness of vessels and slow movement of the blood, while around, and more distant still, there is fulness of the vessels, with a rapid movement of the blood. From the discrepancy existing among observers regarding the statement as to whether the motion of the blood is slower or quicker when the vessels are contracted or dilated, there is evidence that the contraction alone of a vessel, or its dilatation alone, is not always sufficient to cause the current of blood to be either slow or quick. Other conditions are at work which contribute in no small degree to accelerate or slow the rate of movement in the vessels. Besides the force of the heart's action, there is a mutual relation which subsists between the blood and blood-vessels and surrounding tissue which materially influences the motion of the blood. In the healthy body this mutual functional relation between the minute elements of tissue and the blood is necessary to maintain it in a state fit for transmission through the vessels. The mere contraction of the arteries leading to a part does not tend to stagnation of the blood in the capillaries of the inflamed part; on the contrary, the movement onwards of the blood in the vessels is influenced or modified by the vital functional processes going on between the capillary vessels and the surrounding elements of tissue, and which has been variously named the "capillary force," the "vital force," the "nutritive force;" it is also mainly influenced by the action of the heart itself, and by the physical condition of the vascular tubes through which it has to pass. Accordingly, at first, with contraction of vessels, the current has been described as being quickened. It also sometimes slackens, or even retrogrades for a time, and not unfrequently oscillatory movements may be noticed. But when dilatation is complete, the blood flows with rapidity, and a greater quantity passes during a given time than in the unexcited state of the parts. This is known as the state of "determination of blood to a part," or "active congestion." The natural function of the part thus becomes simply exalted; and it may be said that a step beyond this will pass the confines of that neutral ground which exists between health and disease. With an increased circulation, and such "determination of blood to a part," functional activity is not only maintained, but is promoted and

increased; and unusual transudation of the nutrient material may take place, chiefly of the serum of the blood. Hence the oedema which surrounds an inflamed part. After a time the motion of the blood becomes slower, while the volume propelled is increased, and the retardation gradually increases till the blood-corpuscles are no longer propelled, floating in their *liquor sanguinis*; but accumulating in masses, they advance by a jerking intermittent motion, till at last complete stagnation takes place. The blood-corpuscles now detained exhibit a marked tendency to adhere alike to the walls of the vessels and to each other; thus accumulating together and sticking in the capillaries, while the *liquor sanguinis* flows onwards. To this condition the term "stasis" has been applied. In the immediate neighbourhood, and surrounding the part which is in the condition of *stasis*, the circulation of the blood goes on with increased rapidity: it may even pulsate in the arteries and oscillate in the veins, while it moves with a uniform but rapid flow through highly distended but less turgid vessels. When these conditions exist simultaneously, and the true morbid process is completely established, the capillary vessels may burst, causing hæmorrhage or extravasation into the surrounding tissue, or the serum and *liquor sanguinis* may transude through their walls, without rupture, into the surrounding texture.

The "determination of blood to a part" here noticed, characterized by dilatation of the arteries with increased flow of blood through the capillaries, must be distinguished from the "congestion of inflammation," characterized by the accumulation and stagnation of red and white corpuscles in the vessels, tending to be abnormally adherent to each other and to the vessels. Both of these phenomena, namely, "determination" and "congestion," may result from irritation. The dilatation of the arteries seems to be immediately developed through the medium of the nervous system, while the accumulation of the blood-discs and stagnation of the blood is the immediate and direct result of impaired or suspended function of the minute tissue-elements contiguous to the capillary vessels.

The "determination of blood" and dilatation of the arteries lead to no change in the quality of the blood itself; on the other hand, accumulation and stagnation of blood, in the congestion of an inflamed part, are associated with increased adhesiveness of the red and white discs. Mere determination of blood becomes

obliterated after death by the *post-mortem* contraction of the arteries, whereas the congestion of inflammation is persistent. It is an evidence of organic lesion declaring itself as distinctly in the dead as in the living; and thus the most important, if not the only sign of the early stage of inflammation having occurred during life is recognizable, on dissection, by the intense redness due to the accumulation of red discs adherent to each other in the minutest ramifications of the vessels, and not due to distension of the vessels merely.

Such is a statement of the facts ascertained regarding the early phenomena of the inflammatory process; and they are of such a kind that, with the facilities of study which ought now to be within the reach of every student of medicine, he ought to make such experiments as have been already noticed, or see them made by others, and thus really appreciate the steps of that morbid process which he requires to treat so extensively in practice, and of which he can form but a faint conception from the most lucid description.

II. The constitution of the blood is altered as regards its adaptability to nourish the part.

The nature of this alteration cannot be chemically expressed; but microscopical observation has established a fundamental fact, namely, that the tissues through which the blood flows have such special relations to the living fluid that, in the healthy state, the functional activity of the minute tissue-elements maintains the blood in a state fit for transmission through the blood-vessels; and the first change observed in the blood, subsequent to any impairment of function of tissue-elements, is an increase of adhesiveness of the red as well as of the white corpuscles: but the white corpuscles are now known to be susceptible of much greater adhesiveness than the red; so that slight irritation, leading to impairment of function, causes stagnation of the white sooner than of the red discs. The blood is not thus altered in the first instance throughout its whole mass; but the change is a local one, confined to the seat of the inflammatory process. At one time it was believed that the blood was altered in its constitution chiefly by an increase of the fibrine and the white corpuscles;* but it is now found

* Andral and Gavarret showed that the proportion of fibrine in the blood was augmented in inflammations, when sufficiently severe or extensive to affect the system. In health the average proportion is three parts in 1,000, and in cases of

that the white or rudimental corpuscles of the blood cannot be separated from the fibrine by any known process; consequently the relative amount of fibrine cannot be correctly stated in relation to the blood. And, as in many inflammations these corpuscles are increased, as well as in many conditions, such as pregnancy, in which no inflammatory process exists, the blood is similarly altered, it is not known how much of change is due to fibrine or how much to the white corpuscles. The generation and accumulation of large numbers of white corpuscles in the vessels of an inflamed part is not now received as a fact. The phenomenon may be true as regards some frogs, but not as regards warm-blooded animals; and it is consistent with the experience of three most eminent pathologists who have experimentally examined this subject—namely, Mr. Wharton Jones, Dr. Hughes Bennett, and Mr. Paget—that an especial abundance of white corpuscles in the vessels of an inflamed part is neither a constant nor even a frequent occurrence. Dr. Hughes Bennett's researches relative to leucocythæmia have shown that even the most extreme abundance of white corpuscles in the blood has no tendency either to produce or to aggravate inflammations.

A remarkable phenomenon presented by the red blood-corpuscles in inflammation was first observed in 1827 by Mr. Lister, sen., and by Dr. Hodgkin, and afterwards accurately described by Mr. Wharton Jones.

They observed that when healthy blood is received on a glass plate, or the clean surface of a polished lancet, and immediately examined, the corpuscles lie diffused in the *liquor sanguinis*, but in about half a minute they run together into piles or rouleaux, which arrange themselves in small meshed networks. But if a drop of blood from a patient with acute rheumatism, or with an inflammation, be similarly examined, piles of red corpuscles instantly form, and are clustered into masses, leaving a network with wide interspaces.

This appearance of itself, however, is not a sure sign of inflammation. It may be observed in the blood of the chlorotic female as well as in the pregnant one; in those also in whom a plethoric

severe inflammations it has been found to rise as high as eight, nine, or ten parts in 1,000. This increase commences as soon as the inflammation is established, and ceases when the process begins to decline.

condition as regards the blood exists; in persons in health, whose circulation has been much accelerated, as by violent exercise; and it appears to be the natural state of the blood of horses. It is a phenomenon resulting from an increased tendency to aggregation of the blood-corpuscles, and gives a granular appearance to a thin layer of blood when viewed with the naked eye. When blood is drawn off in quantity, the phenomenon is associated with the formation of what is termed the "buffy coat," as the clustered blood-corpuscles, rapidly sinking, subside to some distance below the surface before the fibrine and the white corpuscles begin to coagulate.

However indefinite and uncertain the changes may be, as observed upon a small portion of the blood, it cannot be doubted that the blood stagnant or retarded in an inflamed part undergoes important alterations; and by a constant succession of such changes the whole fluid may come at length to be materially altered, as indicated by the general effects and constitutional disturbance, extending throughout the nervous and the vascular system, and which may ensue in the train of an inflammation of purely local origin. It is probable that local changes ensue in the blood similar to those we shall have to notice as taking place in the products of growth in and amongst the elements of tissue during the inflammatory process. There is no doubt, as Wharton Jones has shown, that fibrinous coagula occasionally form, and even degenerate, within the blood-vessels. When the stagnation of the blood is not constant, these fibrinous coagula are carried away into the general circulation, giving rise to the phenomena of embolism (to be afterwards described) in the capillary vessels of some of the more solid viscera, such as the brain, lungs, liver, spleen, or kidneys. By the degeneration of such coagula the whole mass of blood may be infected, and constitutional disturbance excited, producing sometimes various and wide-spreading suppuration,—as when purulent infection is consequent on local injury, or when a blood-clot passes upwards, and becoming lodged in the cerebral vessels, induces the state known as softening of the brain.

There are many points or questions deserving of attention regarding the theory of the inflammatory process; but it is also obvious that in a handbook such as this, any mere analysis of speculative doctrines ought not to take up much space. The

following statement will therefore merely embrace as much as possible of those topics of special interest which a more extended and accurate physiological knowledge of the process of inflammation has shown to be the proper objects of more extended inquiry.

In the first place, as to the primary seat of the inflammatory process, there can be little doubt, from the phenomena already described, as well as from the results of dissection, which show the progress and effects of the process, and from the experimental researches of Hunter, Thomson, Wilson Philips, Hughes Bennett, Wharton Jones, John Simon, Paget, Lister, and other observers, that the vital morbid process known as "inflammation" is connected with the minute capillaries, and the most minute elements of tissues which they nourish. Questions relative to the theory of the process are therefore found to be intimately connected with the histological and physiological relations of these parts.

During the earliest period of the process—the period of increment, or of incubation, as it has been termed—it appears to be the inherent properties of the minute component elements of tissues which first undergo a change, and, combined with the reflex actions of the nervous system, seem to maintain, to promote, or to increase the activity of the subsequent stages.

The simplest effects upon the minute elements of tissue, and upon the blood-vessels, are seen to follow the application of the mildest or slightest physical or chemical agents, but which, operating powerfully, are also capable of extinguishing altogether the life of these elements of tissue. When the action induced is mild and gentle, the tissues become incapable of performing their wonted functions; and, provided the mechanical or chemical agency has not been too severe, the impairment of function may subside, and the tissues will return to their normal state of functional activity. This is "Resolution" of the inflammation.

Such irritant causes acting either immediately from without, or through the blood, or through the instrumentality of the nerves, each component texture of the part becomes affected as soon as it is brought in contact with the irritant. A gradual contraction of the arteries takes place—the contraction following at some interval after the application of the stimulus—is slowly accomplished, and persists for a variable length of time. Relaxation then no less gradually ensues, when the capillaries

open up and slowly dilate till they acquire a size larger than they had previous to the application of the stimulus.

The minute arteries have been shown by the histologist to possess in abundance the structural elements of the non-striated contractile tissue; and in this respect they closely resemble the constitution of the muscular fibre of the intestine. Accordingly, the contractions they undergo have been considered as analogous to *spasms* (as Cullen first suggested); while the succeeding dilatation may be of the nature of *relaxation*, and ultimately of *paralysis*. This paralyzed state is shown from the fact that the same vessels now dilated will not contract upon a re-application of the same stimulus which before made them contract. If the stimulus is made with a needle upon the vessels in the transparent parts of an animal, the needle may be repeatedly drawn over such dilated vessels and no contraction will follow; but with a stronger stimulus, such as that of heat, they may be made to contract again, and even close; and this state of contraction may persist for a whole day, before the vessels again open up and permit the blood to flow (PAGET). On the other hand, the true capillaries seem totally destitute of any structure known to be contractile. They merely consist of a delicate homogeneous membrane, beset with occasional nuclei. A film of collodion is not more homogeneous nor more continuous than the membrane of a capillary (VIRCHOW). Whereas the minute arteries (some of them less even in calibre than capillaries) possess distinct coats, one of them consisting of a single layer of muscular (or contractile) fibre-cells, wound spirally round the internal membrane of the blood-vessel, so as to encircle it from one and a half to two and a half times. The arteries, to their smallest branches, are sometimes contracted to absolute closure, and at other times are widely dilated; whereas the capillaries are never entirely closed, nor do they present any variations in diameter which are not due to the elasticity of their parietes, (LISTER, l. c.)

The most interesting point in the whole process is perhaps that which embraces an inquiry into the cause of the "stasis," or stoppage of the blood, and the exudation of the *liquor sanguinis*, which are the most difficult phenomena to explain consistently with physiology. This is a point which I think the observations of Professor Lister have so very beautifully illus-

trated; but the explanations of other eminent pathologists and experimentalists, if not universally satisfactory, serve to present the subject in a variety of aspects to the mind, which cannot fail to be both interesting and practically instructive. Henle, Simon, Bennett, Williams, Rokitansky, and Paget have all helped to elucidate the process by the following theories:—

The theory of Henle, or, as it is sometimes called, the "neuro-pathological theory," assumes that the stimulus, acting on the sensory nerves of the part, excites in them a state which, being communicated to the spinal nervous centre, is reflected on the vascular nerves, occasions their paralysis, and therewith paralysis also of the contractile coat of the blood-vessels. Various modifications have been made upon this theory; but as the phenomena have been seen to take place in the case of absence of a spinal cord, and in division of the roots of the nerves, and in section of the lumbar and sciatic nerves, such facts are subversive of the hypothesis. Henle considers the stasis as a necessary physical consequence of this dilatation of the blood-vessels, and this stasis, together with the relaxed and dilated state of the vessels, favours the exudation of serum, the consequence of which is, that the plasma of the blood in the part becomes inspissated by a preponderance of albuminoid matter over the salts. This inspissation of the plasma determines endosmotic changes in the red corpuscles, in consequence of which they are disposed to aggregate.

Simon propounds the view that the phenomena are due, not to a reflex action, but to a direct change effected by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.

Bennett ascribes the change as due to a vital force actively operating through the tissues which lie outside the vessels, and which is the only active agency causing the approach of the coloured particles to the capillary walls of the blood-vessels, and the passage through them of exudation.

Paget supposes a mutual relation to exist between the blood, its vessels, and the parts around, which being natural, permits the most easy transit of the blood, but being disturbed, increases the hindrances to its passage.

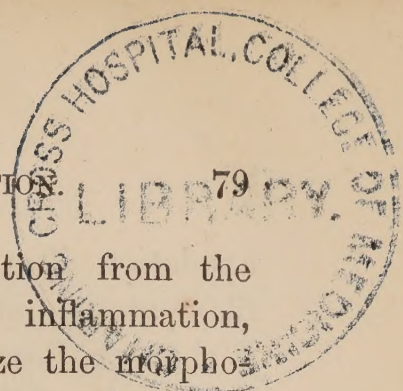
Dr. J. C. B. Williams considers that an essential part of inflammation is the production of numerous white globules in the

inflamed vessels, and that the obstruction of these vessels is mainly due to the adhesive properties of these globules.

Rokitansky is of opinion that the condition of *stasis* proceeds,—1st. From the sticking together of the blood-corpuscles, the heaping up and wedging of them in the capillaries, while the plasma in part flows off towards the veins; 2d. From the inspissation of the plasma, occasioned by the exudation of serum through the dilated and attenuated walls of the vessels, and its saturation with fibrine and albumen; 3d. From the heaping up of the colourless corpuscles, *i. e.*, the nucleus and cell-formations, together with blood-globules; from their sticking together, and from the delicate hyaline, fibrinous coagula which develop themselves among them. Rokitansky considers this to be the most important moment in the inflammatory process, since on the one hand it very specially throws light upon the phenomena of *stasis*, and on the other hand it comprehends the plastic processes which take place in the heaped-up and stagnant blood. It separates in this way the process of inflammation from a merely simple one of exudation. The elementary formations above-mentioned are not merely swept together towards the place of *stasis*, but they originate as new formations in the stagnant blood, which generally presents remarkable alterations.

Wharton Jones describes the progress of *stasis* as consisting,—1st. Of the adhesion of collapsed and dark-red blood-corpuscles to the walls of the vessels; and 2d. The adhesion of other blood-cells to these. The first adhesion of the blood-cells usually takes place at a bifurcation, and the stagnation of blood is seen to begin in those capillaries which are least in the direct course from the artery to the vein, depending in a great measure upon the inspissation of the plasma, or its increased quantity of fibrine and albumen.

Whatever explanation may be given or accepted as to how the phenomena of inflammation in a part are brought about, our views regarding the essential nature of the process have been hitherto modified according as this complex morbid state has been studied by its effects as seen on the dead rather than on the living body. There are some especially eminent pathologists whose combined observations have of late done much to convey a clear notion of the essential nature of this complex process, namely, Alison, Virchow, Bennett, Simon, Goodsir, Redfern, and



Lister. While Dr. Bennett regards an exudation from the blood-vessels as the necessary constituent of inflammation, Alison and Virchow, on the other hand, recognize the morphological changes of the living tissues, such as have been described in inflammation, as betraying merely a *tendency* in a part to such a local change as exudation amongst its structure. That *local tendency* may be so slight that hardly any difference can be appreciated between the healthy changes attendant on normal nutrition, and those changes between the blood and the minute tissues which are of such a kind that a morbid change (inflammation) is established in the elementary components of the tissues themselves, without any appreciable exudation having taken place either amongst the interstices or upon the free surfaces of membranes. To such a condition Virchow gives the name of *parenchymatous inflammation*, meaning thereby that it is a process established locally between the capillaries, the blood, and the component elements of tissue, and expressed by a tendency merely to the effusion from the blood-vessels of such plastic material as may eventually take place.

Inflammation may thus exist as a local morbid process, characterized by an abnormal condition of the nutritive changes between the capillaries, the blood, and the component elements of a texture, without any appreciable exudation. Such an abnormal condition will, under proper regimen and proper remedies, in a case of simple inflammation, seen from the first, completely subside, no interstitial exudation ever taking place.

Examples of this simple form of inflammation have been fully illustrated by Goodsir and Redfern in this country, by their demonstrations of what takes place within the large cells of cartilage. The cells become larger, the number of nuclei increases, and some, or all of them, may undergo fatty metamorphosis under the influence of this the simplest form of inflammation, and which is only manifested by this abnormal nutritive process between the blood and the cells, and which at once leads to these changes *within* the cell-elements of tissue, described by Virchow as a "cloudy swelling" of these parts, *e.g.*, the cells of the uriniferous tubes, and those of the mucous membrane in the state of catarrh. In this abnormal nutritive process, however, there is a constant tendency to the interstitial

exudation of a hyaline material, which may become fibrous or filamentous, and ultimately soft and gelatinous. Virchow, Weber of Bonn, and His, have demonstrated similar changes in the cells of the cornea.

Thus the minute and penetrating observations of Virchow have given a more comprehensive meaning to the process of exudation than it has hitherto, in this country, been understood to signify; and such alterations as he and others have described in the elements of the tissues of an inflamed part have been in a great measure overlooked, except by Dr. Alison and Mr. Simon. The latter especially states that the irritation of the inflammatory process is independent of the nervous influence, *but is a direct change operated by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.* Dr. Alison, also, long ago recognized the tendency to interstitial exudation as attending such vital changes in the constituent elements of a part, and which entitled it to be considered inflamed. The accurate observations of Virchow, Goodsir, and Redfern have shown that such primitive changes do take place before those more palpable phenomena occur which constitute the exudation as described by Bennett, namely, the exudation of decolorized lymph into the interstices between the constituent elements of a texture. Both sets of phenomena alike show that *inflammation is only one of the various shades of deviation from the normal process of nutrition,—a diseased action tending to a local lesion* (*British and Foreign Medico-Chirurgical Review*, January, 1854). That the irritation of inflammation is in some measure independent of the nerves, the following interesting experiment, related and performed by Mr. Simon, may be quoted in proof:—

“A patient had complete anæsthesia of the fifth nerve, dependent (as a *post-mortem* examination subsequently showed) on its organic disease; the conjunctiva, as well as the integument of the face, was utterly insensible: not only was the function of the nerve destroyed, but those reflective nutritive changes of which I have already spoken had taken place, and had exhausted themselves; showing that the nerve was spoiled for participation in the acts of nutrition (whatever they may be) no less than for its more obvious uses as a medium of conscious sensation; the cornea had undergone ulceration, and had healed again. The following experiment was carefully made:—The lids being held

open, a single granule of cayenne pepper was laid upon the insensible conjunctiva; in a few moments it had become the centre of a very distinct circle of increased vascularity, the redness of which slowly became more and more distinct as long as the stimulus was suffered to remain, so that, on its removal, there was a very evident circumscribed erythema on the surface of the membrane. I consider myself justified in believing that this change occurred without any intermediate nervous excitement; not only because the history of the case would lead me to consider the fifth as annihilated; not only because the experiment was totally unattended with sensation; but likewise because there was the very remarkable absence of that sympathetic phenomenon which the faintest remnant of nervous excitability would have produced—namely, there was not the slightest trace of lachrymation” (*Lectures on General Pathology*, p. 76).

Further evidence might be submitted from the papers of Mr. Joseph Lister to the Royal Society, already referred to.

Such being the essential nature of inflammation, it is easy to understand how reasonable is that doctrine which teaches “that the process of inflammation is susceptible, at all times and in all countries, of very great variety as to extent or intensity, and especially as to the constitutional affection associated with it or consequent upon it.”

Products, Effects, or Events of Inflammation.—Care must be taken not to put the *products* of inflammation in place of the *symptoms* of inflammation. When the local impairment of function of the minute elements of tissue in process of inflammation *is confined to a small space, or is carried on upon a minute scale, or rapidly abates*, the inflammation is said to *terminate by resolution* as a general principle; that is, the abnormal action ceases, interstitial exudation does not take place, the tendency to further impairment of function is subdued and passes off, and the part is left apparently as it was before. If, however, interstitial exudation has taken place, and *resolution* is to be effected, the return of the part to health may be followed, for some time, by some impairment of its structure and function.

After the process has thus gone a certain length, an increased local growth of cells, and their liquefaction or reduction to a state capable of absorption (what Dr. Addison calls *cell-therapeutics*), are essential to the restoration of the part. Before the process has attained such a length, however, *resolution* may be simply

effected by a gradual return of all the parts to a natural state: a mere retracing of the steps by which the natural actions had been departed from sufficiently describes the process (PAGET).

The process of *resolution* has been closely watched by Mr. Paget. He has seen, in those cases where impairment of function and actual lesion had taken place, that fragments of fibrine, washed from the blood in the vessels of the injured parts, were borne along and floated in distant vessels. The observations of Dr. Kirkes, also, leave no doubt that similar changes may occur in warm-blooded animals, and may be the source of great evils; may be, indeed, productive of some of those constitutional effects yet to be noticed, by carrying the materials of diseased or degenerate blood from a diseased organ to one that was previously healthy. When the disappearance of the inflammation is unusually sudden and rapid, the event is technically called "*delitescence*," and if at the same time the symptoms of inflammation appear at another part not anatomically connected with the part first diseased, the event is called a "*metastasis*."

When the process does not confine itself to the simple expression of altered nutritive changes between the constituent tissues of a part and the blood; but when the tendency to exudation amongst the interstices of texture continues, and does not subside, as already explained,—namely, by *resolution*,—then it is that (1.) such a material is separated from the blood as will become a medium or *nidus-substance*, in which many changes connected with the growth of new particles, granules, or cell-forms will take place, and the phenomena of which have been so well described by Bennett, Gluge, Paget, Virchow, Beale, and John Simon; and (2.) coincident with this exudation, and the changes which it undergoes, the tissue of the part itself sustains serious alterations. For in all such inflammations especially of the more vascular parts, when there is increased exudation from the blood-vessels, there is a great deterioration of the surrounding elements of tissue. The texture is rendered soft and easily torn, and by such changes of cohesion the elasticity of parts (a circumstance often of very primary importance) becomes greatly altered and impaired.

These changes, therefore, Mr. Paget happily describes as consisting of,—(1.) *Productive* effects—that is, effects resulting from the growth of new particles, granules, or cell-forms, from pre-

existing germinal elements of tissue, and which are susceptible of further development, and also of *degeneration*; (2.) *Destructive* effects, such as *softening*, *degeneration*, *absorption*, *ulceration*, and *death* of tissue.

Productive Effects of Inflammation—Inflammatory Effusions or Exudations.—These consist of,—1. Serum; 2. Blood; 3. Fibrine; and 4. Mucin. These last two are the only *true inflammatory exudations*.

1. *Serous Effusions.*—The effusion of pure serum is said to be very rare. In Inflammation of a serous membrane, as the pleura, the fluid effused is not only greater in quantity than natural, but is also greatly altered in quality. In health the serous secretions are little more than pure aqueous vapour, with a trifling addition of saline matters; but when they occur in an inflamed part, they contain a considerable quantity of albumen, sometimes a portion of fibrine, and at other times the secretion appears to be the pure *liquor sanguinis* which is effused, entirely unchanged in its physical properties. The quantity effused varies, according to the part affected, from perhaps a portion of an ounce to a few pints, or even more than a gallon.

This product of inflammation, but mixed with fibrine, may be seen in the fluid contained in blisters raised by counter-irritants in a healthy person; also in the fluid of *peritonitis*; of *pleurisy* and of *pericarditis*: such also is the fluid that fills the early vesicles of *herpes* and *eczema*, and other cutaneous eruptions. It is also seen in the fluid which surrounds an acute, deep inflammation beneath the skin. The fluid of a common *hydrocele* is another example of serous effusion. The phenomena associated with the production of such a secretion may be often seen surrounding a phlegmon or boil of large size. While the centre or *core* of the boil is hard, it is surrounded by textures into which the *effusion* that has taken place is *serous*. Such serum is seen to a great extent in *pelvic cellulitis* in its first stage. A very demonstrative and interesting example of this is given by Professor Simpson, of Edinburgh, in the *Medical Times and Gazette* for 1859, p. 27, July 9. In such cases the fluid fills the areolar tissue which immediately surrounds the inflamed parts; and when the finger is pressed firmly on the part, the fluid is displaced into the adjoining areolar spaces, which yield to receive it. When the finger is withdrawn, the fluid does not

immediately return, but an impression is left in the shape of a pit. The part which is the seat of *serous* effusion is then said to *pit on pressure*, or to be *œdematous*.

The fibrine of inflammatory serous effusions remains in solution for weeks or months within the body, during life, but will coagulate readily when withdrawn. This delay of the fluid to coagulate within the body is a propitious event. So long as it is liquid, absorption may still ensue without its undergoing any ulterior change when the inflammation subsides. This subsidence of the inflammation, however, is necessary, for it is known that so long as inflammation continues there is impairment of function, and absorption does not take place.

One constant characteristic of the *productive* effects of inflammation is, that growing material is always developed from the pre-existing germinal elements of the tissue involved; and the effusion that results from mere mechanical obstruction to the flow of blood is very different from the fibrinous effusion of an inflammation. In the former case the fluid effused from the blood is merely the serous part, as the fluids of *anasarca* and *ascites*, and will not coagulate. Such fluids, as a rule, neither present fibrine, nor are any granules, particles, or cell-forms developed in them from the elements of surrounding tissue.

Fibrine can only be made to exude upon any surface or part in a state of irritation or inflammation. Such a local change is sufficient to cause the exudation of fibrine, independently of obstruction to the circulation; and the cause of the greatest differences in the nature of exudations is to be found in the special constitution of the irritated parts (VIRCHOW).

It has been clearly shown (SIMON, LEHMANN, BEALE) that there are two essential characteristics of inflammatory effusion,—(1.) It tends to contain certain ingredients in larger proportion than that in which they exist in the blood—excess of chloride of sodium and of phosphates and albumen; (2.) Organic forms find in it a suitable place for growth.

The site of effusion resulting from inflammation is important, as sometimes constituting the chief element of danger—a danger sometimes immediate, from the mechanism of the parts affected. A large quantity of fluid is often poured out in a very short time. The cavity of the pleura may fill in a few hours, and the lung may be compressed by it to a half or third of its

bulk: and if both pleural cavities become thus affected, constituting *double pleurisy*, the patient must die from suffocation, if not at once relieved by allowing free vent to the fluid. This operation is called *tapping* the chest, or, technically, "*paracentesis thoracis*." Serous effusion into the areolar submucous tissue of the glottis may also produce almost immediate death from suffocation, unless the cavity of the larynx is immediately opened to admit the air to the lungs (WATSON).

2. *Blood Effusions or Extravasations*.—Such chiefly occur from rupture of the new vessels developed in the newly formed material, which has just become vascular (ROKITANSKY). In the ordinary course of internal inflammations, extravasations of blood are rare, and betoken an unfavourable state of the constitution generally, such as occurs in typhus fevers, in scurvy, in purpura, or in syphilis. The *post-mortem* evidence of such extravasations is the presence of a coloured cicatrix at the spot of rupture and effusion, and the colour is found to be due to the presence of hæmatoidine, generally in the form of a mass of aggregate crystals, composed of minute rhombic columns, and which may be considered as the regular typical ultimate form into which hæmatine is converted in any part of the body where considerable masses of extravasated blood continue to lie for any length of time, *e. g.*, apoplectic clots and coagula in the Graëfian vesicle of the ovum after menstruation (VIRCHOW).

Mr. Paget correctly observes that we must not confound with hæmorrhages the cases in which the inflammatory products are merely blood-stained, *i. e.*, have acquired a more or less deep tinge of blood, through the oozing of some of its dissolved colouring matter. The natural colour of inflammatory new formations is greyish or yellowish-white, and even when they have become vascular, their opacity in the recent state prevents their having any uniform tint of redness visible to the naked eye. When inflammatory products present the tinge of redness, it is either because of hæmorrhage into them, or because they have imbibed the dissolved colouring matter of the blood; and when this imbibition happens during life, or soon after death, it is important, as implying a cachectic, ill-maintained condition of the blood, in which condition the colouring matter of the corpuscles becomes unnaturally soluble.

3. *Inflammatory Lymph or Fibrine*.—This product is so

named to distinguish it from the lymph in the lymphatic vessels, with which it is probably not identical (PAGET). It is a characteristic primary product of the inflammatory process, and was called originally "*lymph*," or "*coagulable lymph*;" and more recently it has been called "*exudation*," or "*fibrinous*" or "*inflammatory exudation*." "It is at first," says Mr. Paget, "probably always a pellucid liquid exudation which passes through the blood-vessels"—"sweats through them," as Simon hath it,—"and especially through the capillaries of the inflamed part. Its most characteristic general properties are, that it may become a *nidus-substance*, capable of taking some share, or of assisting in promoting the growth of new elements like the natural connective tissue of the body." But the nature of the products of this development in the "*inflammatory lymph*" varies much, according to the part and the state of the constitution; and accordingly Bennett attempts to distinguish the products of *simple exudation* from those of *tuberculous* and *cancerous* exudations. The typical elementary forms which may grow amongst inflammatory lymph vary according to a much greater variety of circumstances. The circumstances which tend to modify the type of the inflammatory process, or impart to it a particular *tendency* in respect to the nature of the productive material, may be shortly stated as follows:—

1. The nature of the tissue in which the inflammation takes place.
2. The period at which the product is examined after exudation has taken place and growth commenced.
3. The state of the blood and the nature of the *zymotic* or *constitutional* morbid state which may be associated with the inflammation.
4. The amount of vascularity which the affected part retains.
5. The amount of the local exudation, and the extent of healthy tissue implicated.
6. The suddenness of the phenomena of exudation and of growth.
7. The persistence of the inflammatory state in its vicinity.
8. The amount of fluidity, serum, serous effusion, blood, or mucus, associated with the *inflammatory lymph*.

These are the chief circumstances which determine and modify the elementary forms which may grow amongst the "*inflam-*

matory lymph," and which may advance to further development, or to degeneration. The circumstances, severally, or more or less collectively, influence the different stages of progression, by which the local lesion of the inflammatory process may ultimately terminate in *resolution*, in *permanent organic mischief*, in *death* of the parts involved, or in *death of the patient*.

There are several typical forms, especially found growing amongst the "inflammatory lymph," and in the growth of which the material of the lymph may take some share—namely, the granular, molecular, or fibrillated development of *fibrinous* products and *corpuscular* forms.

Rokitansky describes these typical forms by the terms "*fibrinous*" and "*croupous*," and Dr. C. J. B. Williams by the names of "*plastic*" and "*aplastic*." Examples of each variety may illustrate the application of the terms. To the *fibrinous* or *plastic* variety belong the serous effusions already referred to, and perhaps also the granular, molecular, or fibrillated growths. The *corpuscular*, *croupous*, or *aplastic* forms of lymph are represented by those growths which never become consolidated, as in the early formed contents of vesicles in *vaccinia* and *herpes*; in the *fluid of blisters* raised in cachectic patients; in some instances of *pneumonia*; and in some forms of inflammation of *serous membranes*. In by far the larger number of inflammatory products these typical forms are mixed in various proportions; and the larger the proportion of corpuscles in new growth, the greater is the probability of suppuration, or of some other degenerative process, and the more tardy is any process of development into tissue, such as that of adhesions, indurations, and the like. In other words, the preponderance of granules, molecules, and fibrillated material in the new growth, is generally characteristic of the "adhesive inflammations;" the preponderance of corpuscles, or their sole existence in a liquid medium, is a general feature of the "suppurative inflammation." The hardness of inflamed parts is due to the former of these typical forms of inflammation, and is exemplified in the case of a *phlegmon* or *boil* before it suppurates; as also in a lung in a state of *hepatization*, when its textures are enclosed by lymph, "just as the stones of a wall are by the cement;" also in the hardening of a chancre.

On the surfaces of inflamed membranes the new growth forms a layer of a membranous firmness or consistence, to which the

name of *false*, *adventitious*, or *pseudo-membrane* has been given. By this new growth the naturally opposed surfaces of parts which are inflamed are apt to adhere. This is commonly seen to be the case between such serous surfaces as the *pleurae*, the *pericardium*, the *peritoneum*, or the *edges of a wound*. The inflammation associated with this organization is sometimes called "*adhesive inflammation*;" and Dr. John Thomson ascertained that this growth and organization might be effected between the surfaces of wounds in less than *four* hours after they were inflicted.

On the surfaces of mucous membranes may generally be seen the "corpuscular" typical form of new growth as a result of inflammation. It has little tendency to cohere, but grows in films, gelatinous masses, shreds, patches, or delicate casts of the surface upon which it was formed. The new growths in chronic catarrh of the intestines are an example; so are the membranes sometimes passed from the cavity of the uterus, and called *dysmenorrheal membranes*. In the "adhesive" form of inflammation the new growth of granules or of molecules may ultimately assume the form of *fibrous* tissue interstitial to the textural elements inflamed. Examples of this organization are seen in the laminated and nodular thickening of the capsules of the spleen, the thickening and induration of the periosteum, or the capsule of the hip joint in chronic arthritis; and by virtue of the peculiar tendency to contraction which fibrinous products possess, the contractions of parts are to be explained which have been the seat of such a form of inflammation.

There are instances also in which the new growth assumes the form of adipose tissue, elastic tissue, and epithelium (KIRKES, VIRCHOW, PAGET); and bone is a very frequent ulterior change which it assumes, especially when the new growth is interstitial to fibrous tissue; but these ulterior events only happen *after* the inflammatory process has ceased in the part. So long as the inflammatory phenomena continue, the tendency of the new growth is to assume the *corpuscular* form, such as pus, rather than the more adhesive forms of organization. Hence rapid organization accompanies, as a general rule, a minor degree of action; and by depressing the action of a part, we tend to prevent the threatened occurrence of suppuration.

The existence of the inflammatory state, associated with an

interstitial exudation, influences the simplest *corpuscular* forms of organization. *Lymph-cells*, or *simple primordial* forms, occur, which are represented by the corpuscles of chyle, lymph, the white corpuscles of the blood, and by those of granulations on the surface of a wound. These simple cells become developed amongst the lymph while it is still fluid, transparent, and apparently homogeneous. The first discernible organic form in the lymph of *herpes*, for example, is that of a mass of soft, colourless, or greyish-white corpuscles, about $\frac{1}{2500}$ th or $\frac{1}{3000}$ th of an inch in diameter, round or oval, pellucid, but appearing, as if through irregularities of its surface, dimly nebulous or wrinkled. It does not look granular, nor is it formed by an aggregation of granules; nor, in its earliest state, can any cell-wall be clearly demonstrated, or any nucleus on adding water. In a few hours, however, a pellucid membrane appears to have grown over its surface, permeable by water, which raises up part of it like a clear vesicle, while the contained mass retreats or subsides to the lower part of the enclosure, and appears more nebulous or grumous than before. A nucleus ultimately forms, and can be distinguished in this mass (PAGET).

From these primordial cell-forms in the lymph either the adhesive or more corpuscular forms of organization may proceed; and all the various forms of corpuscles described by authors as plastic cells, fibre cells, caudate cells, or fibro-plastic cells, and some forms of filaments, are developed from the germinal matter of the surrounding tissue by continuous development. Also, from the development of the primordial granules, corpuscles, or cells, all those elementary forms proceed which are known as "*pus-corpuscles*," "*granule-cells*," "*compound granule-masses or cells*," "*Inflammatory globules*," and much of the *molecular debris-like matter* that makes inflammatory effusions turbid.

The modes of growth as well as of degeneration are well described by Mr. Paget, from whose work on Surgical Pathology the following examples and illustrations are chiefly taken:—

1. The lymph may simply wither or waste, as may be noticed in the vegetations on the valves of the heart or large arteries when they become yellow, stiff, horny, elastic, and nearly transparent; or in the lymph deposited over a compressed lung, associated with empyema or hydrothorax.

2. The fibrine of lymph may undergo changes similar to what is known as fatty degeneration,—changes similar to those which occur in the *primordial lymph-cell* when it is transformed into pus. The two changes generally go on together. To the former change, namely, the fatty-like degeneration of the fibrine, Mr. Paget gives the name of “*liquefactive degeneration*:” the solid fibrine of inflammatory lymph that becomes again liquid when suppuration takes place, as may be observed in a hard mass of inflamed texture when it becomes soft.

This is a degeneration which brings the new growth into a state favourable for its absorption, or to the *resolution* of an inflammation. Examples of such an absorption may be seen in *rheumatic iritis*, and the observations of Dr. Kirkes on the rarity of adhesions of the pericardium in comparison with the frequency of pericarditis may also be explained in this way.

3. Melanic degeneration of lymph and new growths is not unfrequent, as in *peritonitis*.

Concurrent with these degenerations of the lymph-granules and molecules are the degenerations of the *corpuscular elements*.

1. They may wither, as in the dried-up pus of chronic abscesses.

2. The fatty degeneration of cells is said to be shown in their transition to the *granule-cell*, known also as the *inflammatory globule* of Gluge, or the *exudation-corpuscle* of Bennett. The history of the formation or growth of these corpuscles is still doubtful.

The description of them, as originally given by Gluge, in describing the alterations of blood in inflamed parts, is as follows:—

He observes “that the blood-globules lose their tegument and their colour. Their inner substance alone remains, which, however, does not remain solitary; but by means of a whitish connecting material the masses become agglomerated, and form dense, opaque, round groups, containing on an average from twenty to thirty of the smaller bodies, which, examined singly, are quite light and transparent. By means of pressure or acetic acid the associated granules break down into the individual bodies, and we see that the opacity is merely owing to the association. The associated bodies have a diameter in the mass of from $\frac{1}{20}$ th to $\frac{1}{30}$ th of a millimetre; the single granules are from $\frac{1}{500}$ th to $\frac{1}{400}$ th of a millimetre. These associated bodies,” says Gluge, “I have seen in

the blood-vessels, so that we have not here to do with a fluid which, transuding through the coats of the blood-vessels, is changed into granules. They escape by bursting the capillaries."

That this cell or corpuscle is formed within as well as without the blood-vessels, is apparent from an examination of inflammatory lungs or brain-substance. The corpuscles may be seen to coat the blood-vessels exteriorly and interiorly to their walls; and the formation of the corpuscle of Gluge can also be traced through stages of development, as described by Vogel, Bennett, Kölliker, Hasse, and myself; as well as through stages of degeneration from the normal state of some corpuscular elements (textural or morbid), the occurrence of which has been described by Reinhardt, Dr. Andrew Clark, Dr. W. T. Gairdner, and Mr. Paget.

The essential ingredient of which the compound granule-cell is composed appears to be oily or fatty matter; and these cells vary considerably in their appearance, according to the fineness with which this matter is divided. In some the oil-drops are large, in others they are small and quite granular. They are by no means confined to inflammatory parts. Kölliker, in examining morbid products in an animal, has seen oval blood-discs included in these corpuscles, showing that the cell-membrane may be in some instances a subsequent formation in their progressive development. This view of their nature would imply that a number of the original oil or fluid granules come into contact with each other, and cohere into a glomerulus, which subsequently becomes invested with a membrane, and constitutes a cell, the contents of which gradually undergo some morphological process by which they are resolved, and ultimately pass into the circulation (SIMON).

My own observations on this point, published in 1849, and chiefly made upon inflamed pulmonary tissue, led me to express the same result, as to the nature of this compound corpuscular development, in the following statement:—

"1. The formation of clear, transparent, non-nucleated cells may be observed.

"2. The formation of cells with a nucleus and nucleolus are seen, differing from pus-corpuscles in their large size, and in having a single nucleus. These are formed in the fluid of coagulated exuded matter,

and become gradually filled by minute granules, which, when few in number, readily admit of the nucleus being seen. Subsequently, however, they conceal it; and the originally smooth cell-membrane becomes rugged, the granular cell appearing as a spherical agglomeration of granules. Subsequently the cell-wall appears to vanish, the enclosed granules to separate from one another, and to fall into irregular heaps" (*Edin. Med. Journal*, No. 178, for 1849).

The following are the general facts connected with the appearance of these corpuscles:—

"1. They are formed in greatest abundance during the first stage of the exudation (the second stage of pneumonia, according to Laennec).

"2. As long as the capillary circulation is going on, and before complete stagnation has taken place.

"3. When the redness and condensation is the greatest, the corpuscles begin to disappear, or are not seen.

"4. They disappear altogether as the red softening passes into grey, becoming liquid.

"5. They are imperfectly formed, or not at all, in the deposits that occur during the progress of typhus fever or typhoid fever."

Associating these observations with the descriptions of Mr. Paget relative to the liquefaction of fibrine;—with those also of Zwicky and Gulliver, who found these corpuscles in the softened apex or centre of arterial clots;—with those of Simon, who states that they are often found in the fibrinous clots of veins;—with their occurrence in the mammary secretion, in the softened parts of encephaloid cancer, in the vicinity of apoplectic effusions, and that generally they are extremely apt to be present where blood, or the products of exudation or secretion, are undergoing absorption;—does it not appear probable, moreover, from the lucid description given by Mr. Paget (when he says, that during the formation of these corpuscles "they present a gradual increase of shining black-edged particles, like minute oil-drops, which accumulate in the cell-cavity, and increase in number, and sometimes in size also, till they fill it") that these compound granular cells, when associated with inflammatory products, fulfil a very important function, as the media through which the liquefied, softened, and disintegrated products of inflammation are gradually absorbed?

The observations of Reinhardt, Dr. Andrew Clark, Paget, and

Gairdner also place it beyond a doubt, that compound granular cells may result from a fatty degeneration of the textural cells of a part; just as calcareous or pigmental degenerations occur, and which are common to primordial cells. While there can be no doubt, therefore, that fatty degeneration of lymph or textural elements may lead to the appearance of compound granular cells, that process can scarcely be called degeneration which is associated with development, growth, and complete absorption, by which the indurated and confused parts of an inflammation, such as the solidified portions of a lung in pneumonia, are ultimately cleared up.

Degenerate products are usually persistent, but the compound granule-cell is not. It seems to have an important function to perform in the removal of fluid, effete, or softened exudations, after which it too disappears.

The most frequent and important result of inflammation is the formation of pus by the growth of pus-cells. If a phlegmon or boil be observed, when it is a firm, hard, and solid mass of texture and exudation, we may feel in a few days that the solid mass has become fluid, and that it has not increased in bulk. The solidity and hardness are due to the inflammatory changes and effusion, the softening is due to the growth of pus-cells developed from the germinal elements of surrounding tissue (VIRCHOW, BEALE). So it is with the cells of vesicular eruptions which become pustular. The new cells there also become pus-cells—a change which may be accomplished in twelve hours, or sooner (PAGET). The following circumstances point to the development of pus from pre-existing germinal matter, namely, that,—(1.) A preliminary lymph-cell cannot always be discerned; (2.) The modification of the suppurative process, which occurs in the inflammation of mucous surfaces, where the formation of pus seems at once to take the place of the natural cell-growth, without any apparent distinction or alteration of the membranes of the mucous cells, corresponding in this instance to the most simple idea one can have of what Virchow terms *parenchymatous inflammation*, as described at page 79. Ultimately the natural mucous secretion undergoes a change. The characteristic cells on its surface drop off in all stages of abortion. Impaired cohesion of parts results,—an invariable expression of the inflammatory tendency. The epi-

thelial covering becomes less characteristic, and gradually declines to small and simple cells, mingled with many primordial cells, which appear to have been hurried from the surface before they had time to undergo their legitimate development into the perfect mucous cells. From this sketch of what occurs, "it will be obvious," as Mr. Simon writes, "that the anatomical distinction between pus and mucus must be as useless as the so-called chemical tests. Infinite gradations between the two destroy all practical value in such criteria. Mucus, *as a copious fluid secretion*, has no existence in health: the only natural secretion of a mucous membrane is its epithelium, which ought not to exist in quantity sufficient for any evident discharge. If the secretion be hurried, it immediately begins to assume the forms and physical characters of pus, even to the splitting of its nuclei with acetic acid." In short, the essential process of inflammation has been established *in the very cell itself*, by the abnormal nutritive morphological relations which take place between it and the blood in the processes of life.

Inflammations of mucous membrane with a *mucinous* exudation (quite as characteristic of inflammation as *fibrinous* exudations) appertain to certain organs, *e. g.*, the gastric catarrhal inflammations. Such mucus is loaded with *mucin*, as a characteristic product of the inflamed mucous membrane, and which gives the tenacious, stringy character to the discharge.

Between healthy pus and healthy mucus there can thus be no confusion; but there are conditions between the two which yield neither "*praiseworthy*" pus nor healthy mucus.

Formation of Pus—Suppuration.—Well-formed, perfectly elaborated pus is a smooth, viscid, yellowish or cream-coloured fluid, specifically heavier than water, averaging generally about 1.030, having little or no smell, and of an alkaline re-action. Microscopically, it is seen to be composed of certain essential constituents, namely, the pus-cell, and often minute clear particles, which seem to have some relation to the pus-cells as rudiments or nuclei of them. These constituents float in a fluid or serum called the *liquor puris*. The *pus-cells* are about $\frac{1}{25000}$ th to $\frac{1}{30000}$ th of an inch in diameter, pellucid, filled with semi-fluid albuminous contents, and sometimes containing a few minute oil-globules, which give the cells a granular appearance. Their shape appears to depend upon the density of the *liquor puris*. Sometimes a

distinct, circular, dark-edged nucleus may be seen in the paler corpuscles, and sometimes two, or even three particles, like a divided nucleus. The minute clear particles often seen are not more than $\frac{1}{100000}$ th of an inch in size. Such are the components of *good, healthy, or praiseworthy* pus—the *pus laudabile* of the older authors—literally, the *pus to be commended*, as showing a benign form of inflammation, indicating that the process, though a morbid one—a disease—is going on regularly, and promises a fortunate issue (WATSON). It is the *laudable pus* of surgical writers. When, however, the process deviates from the state of health—deviates from the usual and regular course of the morbid action in a person otherwise healthy—then we find not only variations in the pus-cells, but multiform mixtures of withered cells appear, with molecular and fatty matter, escaped and shrivelled nuclei, blood-corpuscles, and fragments of granular matter like shreds of fibrine. The *liquor puris* becomes unduly liquid, and the pus is then said to be *watery* or *ichorous*. It may even, in weak and tuberculous patients, consist chiefly of a thin serum, mixed with flakes or curdled, when it has been called *serous pus*. When the colouring matter of blood is mixed with it, it is called *sanious pus*. Chemical or vital changes of various kinds bring about a peculiar decomposition in pus while yet in contact with living parts, although it is probable that atmospheric air, or gases from an internal cavity, may have to do with the change; but hydrosulphate of ammonia is frequently developed, especially in abscesses about the alimentary canal, near the tonsils or the rectum. The stench is then most offensive when the fluid is set free. Pus, besides possessing certain chemical properties, may possess certain specific properties: thus it may be impregnated with certain poisons, as that of syphilis, or of small-pox; it is also often, in certain constitutional states, loaded with foreign matters, such as urate of soda.

The formation of pus is termed *suppuration*. It takes place under three conditions, namely,—(1.) Circumscribed; (2.) Diffused; and (3.) Superficial.

As examples of the circumscribed formation of pus may be mentioned an *abscess*, a *boil*, or *phlegmon*, in which the suppuration is enclosed within a cavity whose walls are composed of connective areolar tissue, and into which interstitial exudation

of inflammatory lymph and serum has extended over a certain area. It happens that while the central portion of an area has become purulent (*i.e.*, has produced pus-cells as a result of the continuous premature proliferation of tissue), the peripheral part has maintained its firmness and solidity by activity of nuclear growth—and sometimes a “thin, opaque, yellowish-white layer, easily detached,” separates the suppuration area from the denser part. This has been called a “*pyogenic membrane*,” from the supposition that its function is to secrete the pus, whereas the nuclei and cells of the denser part are growing by continuous but premature development into pus-cells. Abscesses are sometimes formed without any of the usual accompanying signs of inflammation being present. They are generally slowly formed, and are named *old* or *chronic* abscesses. When suppuration happens in the natural cavities of the body it is still circumscribed. It is not then, however, called an *abscess*, but a *purulent effusion*.

Diffuse suppuration is exemplified in *phlegmonous erysipelas*, or the *purulent infiltration* of an organ. In such cases the inflammation extends through a wide extent of tissue, and from first to last the boundaries are ill-defined. The growth of pus-cells is distinctly interstitial. They are generally rapidly formed, and the tissue becomes thoroughly infiltrated, as if soaked in pus. The usual want of cohesion in the elements of tissue involved in inflammation prevails from the first, and ultimately large *sloughs*, or death of portions of texture, may take place. In some textures of a loose kind it is believed that the pus may spread about or infiltrate parts by its own gravity, thereby leading to secondary destruction of tissue and the formation of what are called *sinuses*.

The incipient progress of diffuse suppuration is probably not dissimilar to that of a phlegmonous abscess, but the inflammation is generally of a different type, and all the processes are less complete; thus, no fibrinous lymph circumscribes the limits of the abscess, nor does any membrane form to limit the pus. The process of suppuration is less perfect, so that the abscess often contains shreds, or even large portions of mortified and loose connective tissue. The pus is less healthy, is thinner, containing a larger portion of serum, and oftentimes portions of loose fibrinous lymph. The *pointing* of this form of abscess differs also from

that of the phlegmonous abscess, for the pus readily passes from its original seat, by infiltration, and, gravitating towards the most depending position, presents a soft, broad surface, without any indications of *pointing*. Such collections of matter are always of greater extent than phlegmonous abscesses, for the free transmission of pus from part to part occasions a great extension of the original disease. When these diffused abscesses open, the phenomena which result depend very much on the nature of the opening, and how it has been effected. "I have seen," says Mr. Hunter, "large lumbar abscesses open of themselves on the lower part of the loins, which have discharged a large quantity of matter, then close up, then open anew, and so go on for months, without giving rise to any disturbance; but when opened, so as to give a free discharge to the matter, inflammation has immediately succeeded, fever has come on, and, from the situation of the inflamed part, as well as from the extent of the lesion, death in a very few days has been the consequence." The same result has also occurred from liberating collections of the diffuse suppurative process in other parts. In erysipelas, however, which so often gives rise to this form of abscess, a free opening is often necessary, to allow of the escape of the portions of loose areolar tissue they contain.

Superficial suppuration may be observed in *gonorrhœa*, *purulent ophthalmia*, and generally in *inflammation of mucous and cutaneous surfaces*; and the growth of pus can be clearly traced where stratified, as well as columnar, epithelium naturally exists. Upon the skin the development of pus may be seen to proceed from the *rete Malpighii*, as a growth by continuous premature development of cells from this part of the young cuticle. In proportion as these young cells give birth to younger germs (proliferate), a separation of the harder layer of epidermis ensues, and a vesicle or pustule is the result. The exact spot where the growth of pus occurs corresponds to what would be the superficial layer of the *rete Malpighii*; and if the membrane of the vesicle be stripped off, the cells of the *rete*, in process of conversion into pus, in place of epithelium, will adhere to the epidermis, and be stripped off with it (VIRCHOW). In the deeper layers the cell-elements, which originally have only single nuclei (centres of nutrition, growing or germinal centres), divide, so that their nuclei (or centres of growth) become more abundant.

Single cells have their places taken by several, which, in their turn, again provide themselves with dividing nuclei, and so the process of multiplication goes on.

Dr. D. R. Haldane, of Edinburgh, has observed and recorded the continuous development of pus-cells from the cylindrical variety of epithelium. In a case of small-pox he found the larynx and trachea coated over with a soft, dirty-looking deposit, which was found to consist of pus-cells. On gently scraping the surface, the cells were found enlarged, and, in place of containing a single nucleus, each contained several—three, four, or more. These were derived from the proliferation of the original nucleus. External to the cells were young ones in all stages of development (*Edinburgh Medical Journal*, Nov., 1862, p. 439).

The more completely the epithelium is of the stratified kind, the less is the surface liable to ulceration (*e. g.*, the urethra in gonorrhœa); but those mucous surfaces where the epithelium is of the cylindrical form scarcely ever produce pus without ulceration (*e. g.*, the intestines). Pus-cells, mucous cells, and epithelial cells, are now regarded pathologically as equivalent elements, and which may replace one another; but physiologically they are not equivalent elements, inasmuch as they cannot perform each other's functions. Deeply seated pus-formation may proceed from *connective tissue*, or from the nuclei of vessels or sheaths of tissue. An enlargement of the *connective tissue* germs occurs (OTTO WEBER), which divide and subdivide, and so multiply excessively by divisions of the larger germinal masses or cells. Round about the irritated or inflamed parts, where single cells lay, masses or groups of cells are formed, a large new formation grows, and towards the interior of this growth heaps of little cells accumulate. These little accumulations occur at first as diffuse "infiltrations" of roundish masses, encircled by an intermediate growth, which continually liquefies as proliferation of the cells extend. Virchow regards this liquefaction as of a chemical nature; the intermediate substance (which yields gelatine) becomes transformed into mucus, and being ultimately converted into an albuminous fluid, is thus rendered liquid. Thus two different modes of pus-formation are distinguished, according as (1.) the growth of the pus-cells proceeds from the germs of *superficial tissue*, like *epithelium*, or (2.) from *connective tissue*; and two forms of inflammation can in like manner be

separated from each other, namely—(1.) The *parenchymatous inflammation*, where the process runs its course in the interior of the tissue-elements (*e. g.*, connective tissue cells or germ masses, hepatic cells), without our being able to detect the presence of any free fluid which has escaped from the blood, but where softening and fluidity is due to the process above described. (2.) The *secretory* (exudative) *inflammation* of superficial tissue-elements, where an increased escape of fluid takes place from the blood, and conveys the new products of growth and altered secretion along with it to the surface.

The *parenchymatous inflammation* has from its outset a tendency to alter the elements of tissue and their special functions. Whereas the *secretory inflammation*, with a free exudation, in general affords a certain degree of relief to the part. Witness the relief which follows the free flow of mucus in catarrh. It conveys away a great mass of noxious matter, and the part appears to suffer much less than a part which is the seat of a purely *parenchymatous inflammation*. In gonorrhœa also we have an example of how the pus resulting from the *secretory* form of inflammation is carried away by that transudation of fluid (exudation) which removes the pus-cells from the surface, without the slightest appearance of ulceration (VIRCHOW).

The description here given regarding the formation of pus is based on the great fact, demonstrated originally by Goodsir, that all new cells proceed from "centres of nutrition," from other cells, or from the nuclei of them; and, as Dr. Haldane justly observes,—“We must not expect to be able, in the case of every abscess or purulent discharge, to trace thus distinctly (as has been done in the preceding paragraphs) the origin of the pus-cells. There is only a certain stage in pathological as in physiological growth in which the actual mode of development can be followed. We might as well expect to be able to discover, by an examination of the mature foetus, the different steps by which its organs had been formed, as to be able, in a ripe abscess, to determine in what way normal had been converted into abnormal tissues.”

There are especially three events which, with more or less frequency, accompany or follow inflammation in a part. These are *softening*, *ulceration*, and *mortification*.

Softening, or diminished cohesion of tissue, is an almost constant

result. It may be due not merely to mechanical separation by infiltration of the component elements of tissue, but to a loss of the vital cohesive properties, and impaired function of the tissues themselves, which tend towards their liquefaction and degeneration. Examples of this may be seen in the inflammation upon mucous surfaces already referred to, also in the inflammatory red softening of the brain and spinal cord, and in the lungs, where a peculiar brittleness and rottenness is imparted to their fibrous substance or skeleton texture. Such softening is due to vital changes in the proper tissue, often independent of any interstitial infiltration. The most remarkable example of inflammatory softening is that which occurs in bones. An acutely inflamed bone is so soft that it may be cut with a knife (STANLEY, PAGET).

But while some parts are *softened*, others are removed altogether by the process of *interstitial absorption*, as it has been termed. This phenomenon is best seen in bones which have been inflamed. Such absorption gradually precedes the extension of the inflammatory process, and leads, in the case of abscesses, to their spontaneous evacuation, commonly called the "*pointing of an abscess*." The inflammation continues, and the growth of pus moves along in a definite direction, towards the cutaneous or mucous surfaces of the body in its vicinity; but as the integuments are generally the more prone to inflammation, it is probable that they thus become soft, and yield sooner than the mucous surfaces do.

Ulceration goes on in the following way, as seen on an open surface, such as a wound or sore. Three processes progress simultaneously in order to effect ulceration:—(1.) An exudation of *inflammatory lymph* and *serum* surrounds the mass of young cells which constantly continue to grow and to break up (proliferation). (2.) Cells are thus continually growing on the surface, to be carried off by a fresh exudation. (3.) Liquefaction of the gelatinous interstitial material supervenes, and so destruction of tissue takes place continuously. Thus an ulcer forms.

Granulation is one of the modes in which a wound, or sore, or a part previously acutely inflamed, heals. It is then said to do so by "second intention," and is always a reparative process. Granulation may occur with or without suppuration. The first mode is extremely common. The latter is occasionally seen in

the healing of syphilitic maculæ and ulcers of the cornea, and Mr. Hunter conceives he once met with it in the union of a broken thigh bone.

Granulation is associated with an exudation of inflammatory lymph, into which old vessels extend, and new ones are formed. A new surface thus results, which is "granular"—the granule being a small conical tumor or growth, composed of a mesh of terminal loops, formed by capillary vessels shooting into the effused lymph. The figure and colour of the granulation are determined by the state of the circulation; when that is feeble and inclined to stagnate, the granulation is broad, flat, and spongy, and either pale or of a livid hue; when, on the contrary, it is vigorous, the granulation is conical or acuminate, and of a bright-red tint (TRAVERS). The vessels prolonged into the granulation are more or less tortuous, and so numerous as to require a high magnifying power to exhibit their distinctness after successful injection. These vessels become contracted to obliteration as the period of cicatrization approaches. Granulation may take place from a surface, or from the sides of an abscess. If from the cutaneous tissue, the sore heals by a process of skinning; the skin always springing from the edges of the wound. Again, if granulations spring from the walls of an abscess, their opposite surfaces may unite. Granulations sometimes form with great rapidity. Mr. Hunter has seen, after trephining a patient, the dura mater strongly united to the scalp in twenty-four hours. Granulations, however, have not in all cases an equal disposition to unite. Thus the granulations of fistulous abscesses are little prone to adhere, their surfaces being often as difficult to unite as those of a mucous membrane; indeed, it is often impossible to produce adhesion except by exciting a considerable inflammation. A part having healed by granulation, uniformly contracts. This contractile force is so great that although the sore made by the amputation of a thigh is seldom less than seven or eight inches in diameter, yet the cicatrix left on healing is hardly more than an inch or an inch and a half. From this cause we find, in parts that have been the seat of abscess, a marked depression at the point of cicatrization.

The reproductive energy of parts which heal by granulation, however, is not great. It is rare that the original tissue is perfectly reproduced. No fat, for instance, is re-generated in

ulcerated adipose tissues; a muscle being divided, unites by a cicatrix of connective tissue, no muscular fibre being reproduced; and a divided cartilage unites by tough fibrous tissue, but not by a cartilaginous bond of union. The skin, when destroyed, may be reproduced as a good imitation, yet generally it is imperfect. After small-pox, the *rete mucosum* is either slow in forming or never forms at all, so that the cicatrix or *pit* remains whiter than natural. Neither the smooth muscular fibres, nor any of the glandular structures of the skin are formed in its scars; but its fibro-areolar and elastic tissues, its papillæ, and epidermis, are all well formed in them. The reparation of the mucous membrane is equally imperfect, the villi being always wanting. The reparation of a flat bone, such as the cranium, is so slow that ten, twenty, and even fifty years pass away before a small trephine hole is filled up with bony matter. In like manner a healed cavity of the lungs is always marked by a cicatrix of areolar tissue altogether different from the original structure; neither, as far as we know, is the proper tissue of the liver, of the spleen, or of the kidney restored. A nerve simply divided is united by nervous matter in about twelve months or more; and the union is quicker and better in all tissues if air is excluded from the healing of the part.

It is a rule of all cicatrices, that the newly formed part is harder and of greater density than the original structure. Muscle, for instance, unites by coarse, dense, connective tissue; tendon most frequently by a harder and less pliant, but not tougher tissue, and sometimes by bone; and bone after a fracture is a more compact substance, and contains more phosphate of lime than before the accident; but, notwithstanding this addition, the new bond of union is not so strong, nor the living actions so energetic, as in the original structure. For when the constitution becomes enfeebled by severe disease, of a scorbutic kind especially, an old sore has been known to open, and the ends of a once-broken bone again to separate. It is equally a rule that a part having been once inflamed, the liability of the part to that form of inflammation is greatly increased; and also when new membranes or tissues have formed, that these tissues are infinitely more prone to disease than the original membrane.

Mortification is the death of a part, and may be complete or incomplete. In the soft parts the former is termed *sphacelus*,

and the latter *gangrene*; while in hard parts, as the bones, there is a somewhat similar distinction, namely, into *caries* and *necrosis*.

Mortification of the soft parts may be white or black in appearance, humid or dry. The mortified part has a black aspect when the blood is extravasated through the walls of the blood-vessels into the affected tissues, giving to the part a purple or dingy hue, while to the touch it is soft, inelastic, and doughy. The mortified part may appear white when, by the action of cold, the blood has been driven from the part, which subsequently freezes perfectly white.

Humid mortification occurs when the blood transudes in a fluid state, and after its exudation separates into its constituent parts, so that the serum, set free, dissolves in it the red globules, raises up the cuticle in bladders, and forms what are termed "*phlyctenæ*." Air, generated by a process of commencing putrefaction, is not unfrequently contained in the *phlyctenæ*, and gives, to the finger touching the part, a sensation of crepitation.

Dry mortification is a rare disease, and has sometimes been caused by the ergot of rye, or other diseased grain, used as food, giving rise to the disease known as *ergotism*. In the year 1716 dry mortification appears to have been to a certain extent epidemic at Orleans, fifty cases having been treated at the Hôtel Dieu of that city. Dodard described it as beginning generally in one or both feet, with pain, redness, and a sensation of heat or burning like that produced by fire. At the end of some days the part became cold, as black as charcoal, and as dry as if it had been passed through fire. Sometimes a line of separation was formed between the dead and the living parts, and the complete separation of the limb was effected by nature alone, and in one case the thigh separated in this manner from the body at the hip joint. In other cases amputation was necessary. Mr. Solly has given an interesting case of this description, which occurred in the practice of Mr. Bayley, of Odiham. The patient was a child three years and seven months old, from whom, by this spontaneous process of nature, both arms were removed above the elbow, the left leg below the middle of the thigh, and the right foot above the ankle joint, being a remarkable instance, in modern times, of this destructive disease (See "*Ergotism*" and *Med.-Chir. Trans.*, vol. xxii., p. 23).

The bones, the brain, the lungs, the liver, the spleen, and the kidney are all liable to *sphacelus* and *gangrene*; so are the different tissues, as the areolar, cutaneous, nervous, and serous. The muscles, tendons, aponeuroses, and blood-vessels are likewise all liable, but in a less degree.

Local and General Symptoms of Inflammation.

Redness, or at least increased afflux of blood, swelling, or at least increased textural productivity, pain, throbbing, increased sensibility, disorder of function, arrest and change of secretion, are the phenomena which are associated with the local morbid state, or with the textures in its immediate vicinity. Increased local heat under all circumstances is constant. This has been recently proved to demonstration by the ingenious experiments of Mr. Simon and his colleague, Dr. Edmund Montgomery (*A System of Surgery*, edited by T. Holmes, M.A., vol. i., p. 42). If the local process of inflammation, however, is carried on upon a minute scale, or in certain tissues, one or other or more of these symptoms may be absent; if, on the other hand, the local process proceeds on an extensive scale, and involves important and delicate textures of vital importance, then we have much more unequivocal expression given, not only to local symptoms, but to complex morbid processes affecting the constitution generally. Of these the chief are:—

I. Inflammatory Fever.—Of the *constitutional symptoms*, as they are termed, the most prominent are those which indicate "*inflammatory fever, symptomatic fever, or sympathetic fever.*" These constitutional symptoms are of the greatest importance, not only by indicating the nature of the disease, as when the inflammation is connected with an internal organ removed from sight and touch; but they are highly important as a guide to treatment. The premonitory symptoms of coldness and shivering are usually very decided, but not of long duration. They are succeeded by a stage of re-action. The pulse is then hard and swift. There is thirst and greatly increased heat of surface. The secretions and the appetite may not at first vary much from the normal state, but on the whole are diminished. Exhaustion and emaciation do not proceed rapidly. This fever is pre-eminently one of strong re-action and vascular excitement, and these characteristics may be said to constitute its *type*.

A most minute description of the disorder of the general frame by *inflammatory fever*, according to its effect on the *systems* of the body, is thus condensed from the account given by the late Professor James Miller (*Principles of Surgery*, p. 39):—

(1.) *The Nervous System.* There are aching, dull pains in the loins and limbs, restlessness, and much discomfort. The will and the power of exertion are diminished. Anxiety or foreboding of evil is felt, and expressed upon the countenance. The head is generally hot, the face flushed, the eyes suffused, and the skin hot and dry. Special sensation is at first exalted, but afterwards the intellectual functions become more and more disturbed. Ultimately delirium is established, and coma may ensue. (2.)

The Vascular System. The pulse ranges from 80 to 130, or more, and the heart's action is proportionally rapid. The pulse is hard, rolling like a cord below the finger, and yielding but little to its pressure; or an irregularity of movement in the artery may exist, and thus a thrill or jar is imparted to the finger. There is increased fulness, as if the vessel were itself enlarged, and held a larger quantity of blood at each impulse; the heart is acting not only more rapidly, but more powerfully than in health; and the circulation is truly accelerated. Frequency, hardness, thrilling, are seldom, if ever, absent; but fulness may be wanting, and the pulse may be small instead of full. This modification is chiefly observed during serious inflammatory action, affecting important internal organs situated in the abdominal region. Hence it is sometimes termed the *abdominal pulse*; the artery resembling a hard, thrilling thread, rather than a cord. This pulse always exists in connection with great nervous depression and debilitated though rapid cardiac action; to which circumstance its smallness is probably due. In affections of the brain, on the other hand, producing coma, the pulse is commonly slow and full; the suspension of cerebral influence appearing to diminish the rapidity, without affecting the force, of the heart's action. There are idiosyncrasies also to be taken into account. The pulse may be naturally slow or rapid—fifty or ninety; and this must be allowed for, when previous inquiry has satisfied us that the patient is the subject of such peculiarity. (3.) *The Respiratory.* Respiration is quickened; the breath is felt to be hotter than usual; and an oppression is complained of in the chest. (4.) *The Digestive.* The tongue may be loaded,

white, and moist; or the edges and central tip may be red and dry: the latter is probably the more frequent combination.

(5.) *The Secerning.* The secretions and excretions in general are materially diminished. The bowels are constipated—mainly from want of mucous secretion from their lining membrane; the skin is hot and dry; the mouth is parched; the urine is scanty, high-coloured, generally acid, sparingly aqueous, and holding much saline matter, with comparatively little urea, in solution.

(6.) *The Nutritive.* Digestion is interrupted; so is assimilation; as the fever advances, so does emaciation; and strength is more and more prostrate.

The chilliness, often amounting to shivering, marks the *date* of the febrile disturbance; and rigors more frequently attend the commencement of spontaneous inflammation than of inflammation caused by external injury.

Regarding the constitutional state characteristic of inflammatory fever, some important general conclusions, especially insisted on by Dr. Alison and Dr. Watson, may be thus shortly stated:—

(1.) It is to be observed that there is no fixed relation between the degree or intensity of internal inflammations and the constitutional fever attending them; nor is the fever always proportioned in its degree of violence to either the size or importance of the part inflamed. In some cases, writes Dr. Alison, where we are sure that we have had inflammation going on under our inspection, to extensive effusion of pus, the pulse has been feeble, the skin cool and damp, and the patient exhausted and faint on the slightest exertion; while in others there is high and more inflammatory fever, and in some of these the organ inflamed has been so to no extent, and its function comparatively little affected, but yet the patient has become comatose nearly as in typhus, and died so. Laennec makes an observation of a similar kind (*Edin. Med. Journal*, May, 1857); and Dr. Watson observes that the fever may be high and very strongly marked in that common complaint, the quinsy, cynanche tonsillaris, or tonsillia, which can scarcely ever be said to imply much danger. (2.) The situation, the extent, and the degree of the local inflammation being the same, the fever commonly runs higher in young and in plethoric persons, and in those of sanguine temperament, than under opposite conditions. (3.)

Inflammatory fever is modified in its expression, and especially in the characters of the pulse, by the nature of the part which is inflamed. This has been already alluded to in regard to inflammations of the abdomen, where the action of the heart is depressed, and the pulse is changed accordingly, tending to death by asthenia; and also in regard to the brain, when the mode of death tends to be by coma, the pulse being slow, laboured, and full. (4.) The type of the inflammatory fever is very much modified by constitutional circumstances, such as the previous habits of the patient, and whether any zymotic disease is associated with the local inflammation. (5.) The inflammatory fever undergoes a further change of type (*a.*) when suppuration takes place; (*b.*) when it continues long; and (*c.*) when mortification or gangrene occurs to a large extent. (6.) The febrile state follows generally the local disease; but (7.) there is also good reason to believe that the *pyrexial* condition, and the condition of *inflammation* in a part, may be excited in some instances conjointly; or, at all events, their periods of commencement may correspond so closely that it is difficult to conceive that one is the effect of the other. Observations are much wanted as to the exact ranges of temperature, as measured by the thermometer, in cases of inflammatory fever, and so to verify or set aside such general statements (See Billroth, in *Year-Book of Syden. Society* for 1861).

When inflammation proceeds to suppuration, a severe paroxysm of shivering is often the first indication of the formation of the pus, and the character of the fever undergoes a great alteration from that just described. The degree of the fever varies greatly even in this case, for a most copious formation of pus may take place from a mucous membrane, as that of the bronchi or urethra, and yet the constitution may hardly suffer in any appreciable degree; while a trifling amount of pus from a serous membrane will often be associated with fever of a fatal character.

In any case the character of a fever depends in a great measure on the constitution of the patient. If that be good, the fever is attended with a white tongue, with little tendency to become brown, also with much heat, and a full, strong pulse. On the contrary, if the patient's constitution be broken or impaired, the fever is of a low type—*asthenic*, as it is called. The event of *suppuration* is generally marked by a *rigor* of greater

or less severity, while the fever hitherto has been *sthenic*. It is the occurrence of the *rigor* in the course of the inflammatory febrile state which gives it prominence and importance. It generally attracts the attention of the patient, and indicates to the physician that *pus* has been produced in the part or organ inflamed. As soon as suppuration is complete, and the abscess ripens, or pus approaches a surface to be discharged, and especially if any important organ is its seat, the fever tends to become *asthenic*, with a brown tongue and a rapid pulse, while the local pain in a great measure subsides. At this period the abscess must open spontaneously, or be opened by art, otherwise the patient may be in danger. The opening of the abscess, though attended with much pain from the contracting of the inflamed walls, is usually followed by relief of all the constitutional symptoms; the pulse rises, the tongue cleans, the appetite returns, and a visible and immediate amendment takes place. If, however, the patient has been exhausted by his sufferings in the earlier stages of the disease, the relief afforded is but transient, the pus degenerates into *sanies*, or is altogether suppressed, fever changes its type, and the patient sinks, too enfeebled to establish the reparatory process.

II. **Typhoid Fever.**—The *type* of fever known by this name is *asthenic* or *adynamic*. Feeble and more feeble the patient becomes; the pulse sinks; there is great impairment of the heart's action, and tendency to collapse; the features become pinched, shrunken, damp, and ghastly; and the skin is covered with a cold and clammy perspiration. Sometimes these *adynamic* characters may pass into that typhoid state in which nervous symptoms, such as delirium, somnolence, and tremors, prevail. These characters are known as *nervous* or *ataxic*. The tongue becomes dry, black, and tremulous, sordes cover the teeth, and harden on the lips and angles of the mouth. Low muttering delirium, stupor, or coma prevail; tremors affect the voluntary muscles, and the *fæces* and urine pass unnoticed. This form of fever sets in as a consequence of some untoward or unhealthy tendency of the inflammatory process, such as when mortification of the part occurs. Any cause, however, by which the system becomes extensively vitiated will bring about this form of fever. It is not necessary that the part should die. Putrescence of the infiltrated exudations in the inflamed part, degenerating and

decomposing, poison the fluids circulating amongst them, and so, by absorption, may induce the typhoid state. If this happens with an internal organ, the event is generally indicated by a sudden cessation of all pain, at which the patient often appears very happy, and even joyous, while to the experienced physician its sudden cessation is assuredly an evil omen (WATSON). The most important vital functions are deeply impaired by a prolonged existence of this type of fever. It tends to death by a complete sinking of the circulation, and diminution and loss of animal heat; or deepening stupor, with oppressed respiration, supervenes; or the patient dies by a combination of both conditions,—*asthenia* and *coma*.

III. **Hectic Fever.**—If suppuration continues beyond the powers of the constitution to supply the process with material to form inflammatory lymph and pus—if the inflammation continues, and becomes chronic as to time, inflammatory lymph continuing to be exuded, and pus continuing to form in profuse quantity, especially if an internal organ is its site—another type of *febrile* symptoms is apt to supervene, constituting *hectic fever*. It is not to be supposed, however, as was once believed and taught, that *hectic fever* is due, in every case in which it occurs, to the continued formation of pus. There are forms of *hectic fever* unconnected with suppuration anywhere, but associated with some analogous wasting of the bodily substance; for example, a prolonged secretion of milk in mothers who suckle their infants beyond the natural period. In all cases where a drain upon the system is established beyond its means, such a complex morbid condition of the body as *hectic fever* may be thus induced, and the mischief may not be revealed by any other symptoms. This type is particularly distinguished from the inflammatory and typhoid forms of fever by its remarkable intermissions, which are usually periodical; a period of remission and a period of exacerbation usually occurring once, and sometimes twice, in the twenty-four hours. It is also characterized by an excessive waste of the tissues of the body; and the sweating which attends the paroxysms causes great exhaustion. The assimilative and nervous functions are comparatively unimpaired, so that it is a febrile state generally of very long continuance. The mind remains perfectly clear—often vigorous and active—even when the body is debilitated; and if the intervals between the paroxysms are

tolerably free from febrile excitement, the *hectic* type of fever may be protracted much beyond what at first sight might appear credible; and thus it is sometimes within our power to alleviate greatly this condition. If, however, the fever does not abate during the remissions of the excessive paroxysm, when sweating continues profuse, and when suppuration or other wasting discharge is excessive, the fatal termination approaches rapidly.

The leading symptoms of this form of fever have been watched and described minutely by many observers, non-professional as well as professional. The *fever* creeps on insidiously, and almost imperceptibly; and the physician is at first led to suspect its existence by a very slightly increased frequency of pulse, and a small degree of heat of skin, occurring generally towards evening, and subsiding before the beginning of the next day. The pulse is subject to temporary quick excitement from slight causes, such as by exertion, by emotion, or by food, as after meals. The heat is especially felt in the palms of the hands and soles of the feet. The excitement of the pulse gradually begins to be more and more easily induced throughout the day; and towards evening the general exacerbation of the febrile state becomes regular, and is unmistakable. *Periodic* exacerbations or febrile paroxysms occur almost invariably towards evening, and remissions now become distinctly marked. The exacerbation reaches its height about midnight, and terminates by a profuse perspiration or sweating stage towards the morning. This sweating is sometimes called *colliquative*, and may be replaced or accompanied by *diarrhœa*. Occasionally a second paroxysm occurs in the morning after breakfast (WOOD), or at noon, as described by Cullen; and as a mid-day meal was common in his day, it is probable that these slighter paroxysms may be attributed to such causes as the simple taking of food. Generally, however, in the earlier periods of this type of fever, the interval from morning till towards the afternoon and evening is free from fever; but in the advanced stage the fever becomes nearly constant, while the evening exacerbations and the morning sweats remain characteristic to the end. The pulse of the *hectic* patient is scarcely ever so hard and full as the pulse in *inflammatory fever*; nor is it so soft and compressible as the pulse of the *typhoid* patient. It expresses a middle condition between the two, of very variable character, both as to quickness and strength,

according to the degree of exhaustion of the patient and the amount of febrile re-action. Often during the paroxysm, or during temporary excitement from slight causes, it reaches 120 beats in the minute, the beat being performed with a jerk, as if the result of irritation upon a weakened heart (WOOD).

The heat of skin during the paroxysm is often considerable, and always distressing, so that little more than the slightest covering can be endured. The respirations are quick and short. The appearance of the face is so characteristic that the *hectic flush* of the cheek, limited to a spot in its centre, is now well known. The delicate bright-red colour and circumscribed form of the flushed spot contrast strongly and often beautifully with the pale cheek, and the bright and sparkling eye with its sclerotic of pearly whiteness. The surface of the skin is harsh and dry, and towards the close of life the region of the ankles are apt to become cedematous. The patient loses flesh rapidly, and as death approaches he becomes exceedingly emaciated. It is then that *diarrhoea* is apt to supervene, and to aggravate the sweating, so as completely to exhaust the remaining strength. The mind, unclouded before, gently wanders now, and the functions of life cease, generally without a struggle. It is often one of the closing symptoms, most strongly marked, in pulmonary consumption; and the non-professional pen of our great novelist, Mr. Charles Dickens, has beautifully portrayed its more striking features in the death of Smike:—

“But there were times, and often too, when the sunken eye was too bright, the hollow cheek too flushed, the breath too thick and heavy in its course, the frame too feeble and exhausted, to escape their regard and notice. There is a dread disease which so prepares its victims, as it were, for death; which so refines it of its grosser aspect, and throws around familiar looks unearthly indications of the coming change,—a dread disease, in which the struggle between soul and body is so gradual, quiet, and solemn, and the result so sure, that day by day and grain by grain the mortal part wastes and withers away, so that the spirit grows light and sanguine with its lightening load; and feeling immortality at hand, deems it but a new term of mortal life,—a disease in which death and life are so strangely blended that death takes the glow and hue of life, and life the gaunt and grisly form of death.”

The forms of fever now noticed, as phenomena which may be

associated with the inflammatory process, are to be regarded as various *types* which the febrile state may assume.

SECTION III.—DEGENERATION OF TISSUE.

Definition.—*Degeneration of tissue implies such a departure from the normal state as would give rise to a palpable appearance in its minute elements of a granular disintegration or detritus; or of any deterioration which, by the functional actions of repair in the normal state, could not have been left there, nor been visible.*

Pathology.—The circumstances under which degenerations occur are of the nature of decay and death. For example, degeneration occurs to an immense extent in the tissues of the aged, especially in the heart and arteries, and to a less extent in the voluntary muscles and the hard textures. Towards the close of the life of a part of the body, degeneration takes place—as, for example, in the textures of the placenta, when utero-gestation is nearly complete. To such degenerations Virchow has given the name of *necrobiosis*, because death and degeneration seem to be brought about by altered life at the close of natural existence. In this respect it may be truly said, that “As we begin to live we begin to die.” A spontaneous wearing out of living parts goes on, so that destruction and annihilation are immediately consequent upon life. *Softening* is the ultimate result of such degeneration, which becomes palpable chiefly by the decided friability of the parts. The minute elements of tissue lose their coherence, and at last really liquefy, so that pulpy or fluid products take their place. When it is remembered, also, how abundantly a granular fatty transformation occurs after death, the nature of degenerations becomes more intelligible; and my friend Dr. Lyons, Professor of Medicine in the Catholic University of Ireland, instituted a series of observations which beautifully demonstrated a process of morphic changes of tissues through dissolution and decay, till the mortal parts of our body return “ashes to ashes” and “dust to dust.” To these morphic changes he has given the name of “*Histolysis*.” To the same end are the demonstrations of Dr. Quain, regarding the conversion of muscle into fat, and of crude flesh generally into *adipocere*, accounting for the enormous fattiness of certain geological strata in which animal remains are abundant (MICHÆLIS, quoted by SIMON). Such

experiments and observations as those of Panum, Melsens, Ascherson, Gluge, Lyons, Simon, Burdach, Wagner, Michaelis, and others, and in which granules, vesicles, and cell-forms appear to arise spontaneously out of homogeneous albuminous fluid, will go far to explain many of the conflicting accounts which are given of the nature of the inflammatory products just described, and of the degenerations. Such forms may undoubtedly arise, as these observers show; and having arisen, they decompose and advance through changes such as Dr. Lyons has described under the name of *histolysis*. On the other hand, the *productive* results of inflammation undoubtedly grow from pre-existing tissue-elements, as already described.

All the degenerations are examples of *atrophy with* changes of texture (PAGET), as distinguished from *atrophy* resulting from simple decrease of bulk, the organ or tissue otherwise retaining its usual form, and to some extent its function. To recognize the following degenerations after death, the employment of the higher powers of the microscope is essential.

(a.) *Fatty Degeneration.*

Amongst the degenerations which are brought about by the spontaneous wearing out of living parts, the most widely spread, and the most important, is unquestionably the *fatty degeneration*. It is attended by a continually increasing accumulation of fat, which replaces the minute elements of tissue in different organs; and Simon concludes generally, regarding the presence of oil or fat in textures uninflamed, that it is essentially a sign of weakness or of death, representing decomposition of effective material. In such *necrobiosis* the elements of the tissue completely perish, and are replaced by fat-granules. Examples of this degeneration may be seen in the minute elements of muscle, especially of the heart; also in the acini of the liver, contiguous to the capillaries into which the branches of the portal vein break up; and in such degeneration the cells ultimately disappear, leading to loss of substance and atrophy of the gland; in the blood-vessels, in the *corpora lutea* of the ovaries, in the renal epithelium, and in many pathological products, such as pus, tubercle, cancer, and the like, when in process of decay; and, in short, in nearly all cell-structures, this degeneration is known to occur, except, perhaps, red blood-corpuscles and the elements of nerve-tissue.

In every texture the degeneration becomes evident in a similar manner. Isolated, extremely minute globules of fat appear in the cell-contents, and, becoming more abundant, they gradually fill up the cell-cavity. Usually the fat-granules appear at some distance from the nucleus; but ultimately they lie as close to each other as in the *colostrum corpuscles* of milk. At last the nucleus is no longer visible, and the membrane of the cell finally disappears—probably by a species of solution. If the degeneration occurs in the more rigid structures, as, for example, in the walls of arteries, the fatty granules retain the form of the cell which they replace. Such degeneration in arteries is first seen in the connective tissue corpuscles composing the innermost layer of the internal coat. Afterwards the intermediate substance softens, the degenerate fat-granule masses fall asunder, and the current of blood may carry away the particles of fat with it. Thus a number of uneven places (cicatricial-like loss of tissue) may be produced upon the surface of the larger vessels without the appearance of ulceration (VIRCHOW).

In fatty degeneration of the substance of the heart there is discoloration of its whole substance. It assumes generally a pale, yellow hue, with peculiar spots on the papillary muscles. Short, yellow streaks, which communicate with each other, are to be seen in the direction of the primitive fasciculi, and pervading the substance of the papillary muscles.

Yellow softening of the brain is a form of fatty degeneration, and this yellowness is due to the accumulation of finely granular fat. At every point where fatty degeneration attains a high pitch, great opacity will always present itself. A transparent part becomes opaque, as in the cornea, where the fatty clouding marks the *arcus senilis*, described by the late Mr. Canton, in persons past middle life, and which has been regarded as an index to the existence of fatty degeneration of other more important organs, although the importance of the sign may have been exaggerated. In some form of Bright's disease the uriniferous tubules become filled with fattily degenerated epithelium, which appears on the surface as opaque spots.

Additional examples of this fatty degeneration are to be seen in the *fatty liver*, and in *mollities ossium*, atrophied renal capsules, and thymus gland, and the muscles—voluntary as well as involuntary—the fatty degenerations of the placenta, of cartilage,

of bone, and of morbid growths: indeed, there is no kind of tissue, healthy or morbid, which may not undergo fatty degeneration.

When the normal structure of the part is thus transformed into fat, it is ultimately destroyed, and the place of the histological elements is gradually occupied by a purely emulsive mass—a kind of milk or fatty *débris*—that is, an amorphous accumulation of fatty particles in a more or less highly albuminous fluid (VIRCHOW).

With reference to fatty *degeneration* in particular organs, see the account given of local diseases.

(b.) *Mineral Degeneration—Petrification.*

The process which is followed by tissues undergoing this form of degeneration is very similar to that described in the previous paragraphs; but it is necessary to distinguish the forms of mineral degeneration as distinct from ossification. Formerly every kind of tissue condensed to the same degree as a bone was considered ossified, and the condition described as “*ossification*.” But although a part may have lime in its intercellular substance, and although stellate cells may be present in it, yet it may be merely “*calcified*,” or, as Virchow terms it, “*petrified*” tissue, and this condition he briefly describes as “*petrification*.” Pathological ossification presupposes that the tissue or part which ossifies is called into existence by growth, and not that a previously existing tissue or part merely assumes the form or hardness of bone by absorbing calcareous salts. Ossification always begins by a growth of new tissue; and deposition of calcareous salts in its substance does not take place till a comparatively late period.

Calcification or *Petrification* is a degeneration comparatively more frequent in the peripheral arteries, and occurs most commonly in cases where there is a tendency to calcifications generally, and where calcareous salts are set free at other points in the system, to circulate with the juices (VIRCHOW). The lesion is to be distinguished also from *atheroma* of the arteries. In both conditions the artery may be felt to be a hard and rigid tube, with a calcareous feel to the knife or the touch. A careful examination microscopically will show that the degeneration is in the middle coat, that *calcification* or *petrification* of the minute muscular cell-elements has taken place, and that the fibre-cells of the circular fibre coat are transformed into calcareous spindle-

shaped bodies. The degeneration may also invade surrounding parts, while the internal coat of the artery may be unchanged. The larger arteries are often brittle, from the mineral degeneration of their tissue—associated often with fatty degeneration (*atheroma*). Patches or plates of the mineral substance may be seen imbedded in the middle coat after the inner membrane is stripped off. When the smaller vessels undergo the mineral degeneration, the deposit resembles particles of oil; and the nature of such an appearance can only be determined by the application of mineral acids, which will dissolve the mineral matter with effervescence.

Nerve-cells, the fibrous membrane of the brain, the pia mater, and the choroid plexus, are all liable to undergo the mineral degeneration. Exudations and new growths are similarly liable. Dr. Bennett has seen the gall-bladder converted into a calcareous shell, and the pericardium into an unyielding box of mineral matter enclosing the heart. The cardiac valves are thus often covered with mineral encrustations. Cancer and tubercle-growths may be transformed by the mineral degeneration; and Dr. Bennett has shown how the calcareous transformation of tubercles is the natural mode of arresting their advance.

The degeneration may follow upon the metastasis of calcareous salts, not excreted by the kidneys, in cases of caries of the bones, necrosis, or osseous cancer. I have seen specimens in the most interesting collection of Professor Virchow which show that metastatic deposits of bone-earth have taken place in the lungs and in the stomach under such circumstances. Considerable portions of the pulmonary tissue were *calcified* or *petrified*, without any apparent injury to the permeability of the respiratory passages. The lesion in the lung looked like a portion of fine bathing sponge. The mucous membrane of the stomach was in like manner transformed into a *calcified* or *petrified* mass. It felt like a rasp, and grated under the knife, so that the stomach-tubes seemed imbedded in a stiffened mass. The basis of such degeneration, in which the lime-salts find a resting-place, are the fine fibrous or connective tissues; and hence the degeneration is seen to occur in fibrous tumors, in serous membranes, in the parenchyma of lungs and stomach (as in the instance just mentioned), in cicatrix tissue on the skin, in the valves of the heart, in the connective tissue of muscle sheath, as well of the heart as

of common muscle; in the tunica albuginea, in the fibrine coagula in the heart's cavities, in aneurismal sacs, and in the thyroid and pineal glands. The cretifications of fibrine, of pus, of tubercle, of cancer, of vegetations, of coagula, all pertain to this form of degeneration; and the process may be traced through all stages of progressive degeneration from the pulp-like condition to cement-like, compact, calculous concretion, as in the phlebolite of veins; also in the turbid chalky, speedily condensing juice of the cysts of the choroid plexus, and the cell-incrustations of the pineal gland concretions, as well as in the calcification of *sarcomata* and cancers. With regard to the degeneration as seen in tumors, Mr. Paget describes two methods by which it advances, namely, a *peripheral* and an *interstitial* calcification. The former is the rarer of the two. In this form of degeneration the fibrous tumor is seen to be coated with a thin, rough, nodulated layer of chalky or bone-like substance. In the interstitial form the degeneration is interspersed throughout the tumor, and so arranged that by maceration a heavy hard mass is obtained, variously knotted and branched, like a lump of hard coral (Paget, *Surgical Pathology*, vol. ii., p. 139).

(c.) *Pigment-Degeneration—Pigmentation.*

In this degeneration *pigment* takes the place of the minute tissue-elements, as fat or lime did in the previously described conditions. It is seen in mucus-corpuscles, as in catarrhal pneumonia, in the pulmonary epithelium, in the acini of the liver, in the epidermic tissue, in the corpuscles of the blood in *ague* and *melanæmia*. As in the former degeneration, so in this one, a distinction must be carefully made between fat-granule cells and pigmentation, for in both cases apparently the same image is offered to view.

The fat-granule cells appear as brownish-yellow corpuscles, but their individual particles have no positive colour; whereas the pigment-cells contain unquestionable grey, brown, or black molecules of pigment, which are opaque (VIRCHOW). The diagnosis between the two is important, as in the brain, for example, where both sorts of granule-cells, namely, pigment cells and fat-cells, may exist side by side. The former points to apoplexy having existed, the pigment originating probably in a solution of the colouring matter of the effused blood, the fat to cerebral

softening. Therefore it is of importance for the pathological interpretation of the diseased condition to distinguish between pigment and fat in the granular form. Such pigment or colouring matter is insoluble in potash and acids—even in nitric acid.

In mucus-corpuscles or catarrhal cells the pigment exists in the form of greyish-black granules. They give rise to the smoky grey spots which are brought up in great quantity in the sputa in catarrhal states of the pulmonary passages; and to an extreme degree where accumulating masses of proliferating epithelium take place, as in catarrhal pneumonia and in the phthisis of colliers, so well described by Dr. Wm. Thomson, (*Med.-Chir. Trans.*, vols. xx. and xxi.)

In the condition known as *melanæmia* (which, like *leukæmia*, has cells circulating in the blood, having made their way into it from definite organs) the cells contain black pigment; in the latter case (*leukæmia*) the cells are colourless. In *melanæmia* coloured elements are met with in the blood which do not belong to it (STIEBEL, VIRCHOW, SCHONLEIN, HEINRICH, MECKEL, FRERICHS, and TIGRI). These pigment-cells in the blood were first seen to occur in the history of melanotic tumors, and were supposed to be due to the passage of particles from the tumors into the blood. This is not yet verified by observation. On the other hand, it is to enlarged spleens pervaded by black pigment that the change in the blood is to be ascribed in such cases, the colour being due to the absorption of coloured particles from the spleen. The class of cases which are the most fruitful source of black pigment in the blood are those of malarious diseases, *e. g.*, *intermittent fevers*, and especially in persons who have been long afflicted with a considerable enlargement of the spleen. In such cases Virchow found in the blood of the heart cells containing such pigment; and the cells that bore the colour resembled in size and form the colourless blood-corpuscles; but there were also other cells of an oblong form and nucleated, within which a greater or less number of large black granules were to be seen. It is in the more severe forms of intermittent fever that such pigment-degeneration occurs. Such pigment is seen to accumulate in the minute capillaries of the brain, attaching to the points of division of the small vessels, and sometimes associated with the comatose and apoplectic forms of intermittent fever. Such pigment is also seen in the

minute hepatic vessels (FRERICHS), where it ultimately gives rise to atrophy of the parenchyma of the liver. In a specimen of liver preserved at the museum of the Military Medical School, a deposition of melanotic pigment in a granular form is visible amongst the interlobular connective tissue, following mainly the course of blood-vessels in an irregular manner; and this case, like all the others yet recorded, was associated with a large black spleen. The contamination of the blood in these cases seems due to a degeneration commencing in the spleen.

In *post-mortem* lesions the textures are thus seen to be very variously tinted, red, yellow, brown, green, or black, generally resulting from chemical alteration in the colouring matter of the blood or bile. The red pigments, as a rule, are due to the altered hæmatine, originally of a yellow colour; and which is the common origin of three different kinds of crystals,—(1.) Crystals of *Hæmatoidine* are the most frequent products of blood-degeneration (VIRCHOW). These are formed spontaneously in the body out of hæmatine; and in their most perfect form present the shape of oblique rhombic columns, of a yellow-red colour, or, in thicker pieces, of a deep ruby-red. In little plates it frequently bears a considerable resemblance to uric acid. In the majority of cases the crystals are of extreme minuteness—difficult to resolve, even with a power of 300 diameters. They are insoluble in alcohol, ether, dilute mineral acids, and alkalies; and exhibit a peculiar play of green, blue, rose-tint, and yellow colours, under the action of concentrated mineral acids. If large masses of extravasated blood continue to lie for any length of time, this is the substance into which the blood is transformed. An apoplectic clot in the brain, for example, is repaired by a large portion of the blood undergoing this transformation, and the colour of the resulting cicatrix is due to the crystals of *hæmatoidine*. When a young woman menstruates, also, the cavity of the Graëfian vesicle, from which the ovum escaped, becomes filled with coagulated blood, and ultimately *hæmatoidine* crystals are the last memorials of the event (VIRCHOW). *Hæmatoidine* is also allied to the colouring matter of the bile.

(2.) Crystals of *Hæmine*, arising out of *hæmatine*, differ from *hæmatoidine* in this, that hitherto they are only known as artificial products which have not yet been seen in the human body. They are of a dark-brown colour. (3.) Rectangular crystals or spicules of *Hæmato-crystalline*.

The yellow pigments are due to blood very much dissolved or dispersed, as in ecchymosis, or to bile, when it is absorbed in the blood and tinges all the textures. Colouring matter due to bile may be recognized in the urine by the play of colours it gives with nitric acid. A small quantity of acid gives a green hue; and, as more acid is added, blue, purple, violet, and a red or brown yellow colour will ultimately appear. Of the brown and dark pigments there are two kinds. One kind loses colour on the addition of nitro-muriatic acid or chlorine water; the other resists not only these agents, but even the action of the blow-pipe. This latter pigment consists of carbon. The former is a peculiar secretion formed within cells, or is a transformation of the colouring matter of the blood (BENNETT). Blue and purple pigments have been seen in urine containing *uroxanthin*, or the *Indican* of Schunk; and illustrate the close connection subsisting between animal and vegetable colouring matters (Parkes *On Urine*, p. 198). For much more interesting observations on the nature of pigmentation, consult Bennett's *Principles and Practice of Medicine*, p. 249.

(d.) *Amyloid or Albuminoid Degeneration.*

This morbid process is one in which the normal textural elements of many organs and tissues are transformed into a peculiar substance, which has suggested, on the one hand, an alliance (in some respects only) with the chemical characters of amyloid compounds, and, on the other hand, with albuminous substances similar to those which pervade the tissues of foetal life.

Professor Virchow, of Berlin, was the first to collect the facts regarding this peculiar form of disease, and to put them prominently forward. He proved the frequent occurrence in the animal economy of a degeneration, distinguished by the production of the peculiar substance to be described, which gradually takes the place of normal elements in the tissues so diseased. But Drs. Gairdner and Sanders, of Edinburgh, had anticipated many of the views and descriptions of the Berlin Professor, and, quite independently of Virchow, they initiated in this country the first steps in the elucidation of this very remarkable degeneration. They showed that the waxy condition of the liver and kidney was due to the same change as that which was seen to take place in the spleen. These valuable communications were made to the *Physiological Society* of Edinburgh; and an account

of them may be read in the *Edinburgh Monthly Journal of Medical Science* for Feb., 1854, p. 186, and also in May of the same year. Notwithstanding these researches, and those of Drs. Harris, Alridge, and others in this country, we have much still to learn regarding, (1.) The conditions under which this degeneration occurs; (2.) The forms in which it exists; and (3.) The symptoms of the lesion.

This degeneration has been long known by a variety of names. For many years the morbid anatomist has been familiar with a "bacon-like," or "lardaceous" infiltration of several solid organs of the body, and especially of the spleen and the liver. Portal and Abercrombie described the morbid condition in the liver as a "*lardaceous degeneration*;" and Hodgkin and Bright described the same disease as an "*albuminous infiltration*." In 1842 Rokitansky was the first to give a clear account, and to describe in detail the "*lardaceous*" infiltration of the kidney with an "*albuminous*" transparent substance. The lesion so described constitutes his eighth form of "Bright's disease." But Rokitansky made no chemical examination of the infiltrated material. He simply assumed, from its general appearance, that it was of an *albuminous* nature, and he rightly recognized its pathogenetic relations to certain cachexiæ. Budd has described the disease as "*scrofulous enlargement of the liver*." Oppolzer and Schrant have described the lesion by the name of "*colloid*," and Baron by the name of "*carnification*." The pathologists of this country have hitherto described organs so diseased under the term of "*waxy degeneration*."

Such are the names, derived from appearances generally, under which the peculiar degeneration has been described before microscopic examination demonstrated the structures implicated.

Chemistry and micro-chemical investigations have modified the views regarding the nature of the disease, and now and then have led to modifications in the nomenclature. Under this kind of inquisitive investigation it has been described, (1.) By Virchow under the name of "*animal amyloid*," he believing, from the behaviour of the transformed substance with iodine and sulphuric acid, that the substance must be classified with the vegetable carbo-hydrogens—cellulose and starch. (2.) Meckel retains the name of "*lardaceous*" or "*cholesterine disease*," believing that the essential character of the degeneration consists in the develop-

ment of a peculiar fatty or lardaceous matter, of the nature of cholesterine. (3.) The more extended and definite examinations by Friedreich and Kekulé have shown that the substance of the purest amyloid degeneration more closely resembles the *albuminous principles* than any other substance we know of; and (4.) Schmidt has arrived at the same conclusion. The question, therefore, is not yet definitively settled as to the exact nature of the substance into which the tissues are transformed in the so-called *amyloid degeneration*, but the weight of evidence points to its being *albumen* in some form; and the albuminoid deposits in the spleen of children, so well described by Dr. Jenner, must be classed as examples of this degeneration, and probably also the special lesions in *rickets*.

Investigations relating to amyloid degenerations have taken especially two directions. Pathologists have endeavoured, (1.) To trace the extension of the process of degeneration throughout various tissues and organs of the body; (2.) To determine the essential nature of the material into which the tissue is converted.

It was Professor Virchow who first turned the inquiry into the direction it has now taken, and which has given a remarkable interest to the micro-chemical investigation of the substance into which the minute elements of the tissues and organs are transformed in amyloid degeneration. Virchow stated that the large Malpighian sacculi in the spleen (which, in some instances, looked like boiled grains of sago) were sometimes composed of a substance which gave the chemical re-actions of cellulose, as seen in plants. Cellulose and starch are both vegetable constituents—"isomeric" forms of some common material; and what gave special interest to the observation of Virchow was the discovery that cellulose is also an element in the covering or skin of the "Tunicata"—a genus of acephalous mollusca—and therefore not a constituent of only vegetable organization.

This discovery of cellulose in animal tissue induced Virchow to look for it or its analogue—namely, "starch"—in the human subject. He recognized it in the *corpora amylacea* of the brain. These contain a substance chemically related to starch or cellulose; and these bodies were first seen and named by Purkinje, who gave them the name they have, not on account of chemical characters, but because he observed them to be laminated like starch. Of these *corpora amylacea* there are two kinds, namely,

—(1.) Mineral bodies with concentric circles more or less soluble in mineral acids; (2.) Others which assume a blue tint with iodine, and a violet colour on the subsequent addition of sulphuric acid. The relations of these two kinds to each other are still unknown. The first are the calcareous particles known as brain-sand; and *both* were at first described under the name of "*corpora amylacea*" by Virchow, which has led to some confusion. The term ought to be restricted to those bodies which, by physical and chemical characters, are assimilated to starch. The mineral bodies erroneously described as *corpora amylacea* are chiefly found in the cysts of the choroid plexus and in the pineal gland. On the other hand, the starch-like bodies have been found by Virchow, Rokitansky, Scherer, Kölliker, Busk, and other observers, in the *ependyma* of the ventricles, the *septum lucidum*, the *fornix*, the *auditory* and the *optic nerves*, and also in the *prostatic* ducts. Concentric lamination of these bodies is not always present; nor is the re-action with iodine and sulphuric acid constant. For these reasons Virchow began to examine those organs whose morbid state was described by the names already mentioned as having been given to the fatty or waxy spleen. He applied solutions of nitric acid, which, when hot, gave a yellow hue; he applied caustic ammonia, which gave a brown colour; and from behaviour with re-agents generally, he concluded that the substance was "ALBUMINOID" in its nature. Iodine and sulphuric acid were subsequently tried. Iodine alone gave a strong yellow-red; sulphuric acid being added, developed a blue colour, passing into a strong violet hue. An excess of acid destroyed the violet hue, causing a dark brown-red colour, passing into yellow. Meckel, subsequently to these observations of Virchow, came to the conclusion that there were four forms of this waxy material—that the basis of them all was a peculiar fat allied to cholesterine rather than to starch—that various saponaceous products are formed, ending in the development of cholesterine; and although he did not sustain his statement by anything like sufficient proof, he made the important discovery that it was the system of small arteries and capillaries which first suffered in this degeneration.

The inquiry into the chemical nature of the lesion becomes still more interesting when connected with the observations and discoveries of Bernard, Pavy, and others, on the "sugar-produc-

ing" functions of the liver, and on the material so formed, which may be separated by chemical processes, and has been recently shown by Dr. Robert M'Donnell to be a substance which enters largely into the constitution of most of the tissues of the embryo (*Proceed. Royal Society*, vol. xii., p. 476). The results of these inquiries bring the "starchy substances" of animals in very close physiological alliance. The material so found has been called indifferently, "glycogene," "amyloid matter," "zoo-amyline," or "animal starch." It owes its origin, not to any direct function of the organ, but its formation seems to take place almost immediately upon contact with albuminous matter, when this remarkable product is the result, and which may be obtained as a white powder. It seems capable of being produced in greatest abundance by the hepatic tissue; but its formation may proceed at any part of the vascular capillary system. If, therefore, it is thus formed normally, it may also be formed, retained, or transformed in a morbid way. In *diabetes* we have an instance of the transformation of the product into sugar at the expense of the tissues at large; and which is so discharged by the urine.

The amyloid degeneration we are now considering has thus had various names to denote its presumed chemical nature, namely,—(1.) *Cellulose degeneration*; (2.) *Amyloid degeneration*; (3.) *Cholesterine disease*; and now (4.) *Albuminoid degeneration*.

The analysis of the pure matter is very defective. Such as it is, it shows the substance to be *albuminoid*, and combined with *nitrogen* rather than *starch*; and those who describe the re-action of *cellulose* and *starch* with iodine and sulphuric acid, seem only to agree with each other in giving singularly diversified descriptions of colour, which, perhaps, to those familiar with the writings of the late Dr. George Wilson, on colour-blindness, may be accounted for. Such diversity may be explained in some measure, also, by the fact that the degree of concentration of the re-agents materially concerns the results; for, as Virchow correctly observes, the blue coloration is only got after a considerable period, and in practised hands, and it may pass from a bright purple to a very blue colour. Nevertheless, the action of iodine solution on the so-called amyloid tissue is peculiar and definite, independently of a blue colour.

The appearance of a chemical re-action, which gives a hue

different from the mere dyeing with the iodine, and which *suddenly* deepens in tone, from the moment it begins to take effect, to a deep brown-red colour, is sufficiently characteristic. When this takes place with the solution of iodine ALONE, it distinguishes at once the substance from *cellulose* and *cholesterine*. The following tabular statement will show the differences more clearly:—

CHOLESTERINE.	AMYLOID OR ALBUMINOID.
1. Unchanged in colour by iodine alone.	1. Immediate coloration (of the nature of a re-action) by iodine alone.
2. Insoluble in water.	2. Dissolves in warm or boiling water. (Boil sections in a test tube.)
3. Melts with heat.	3. Does not melt with heat—only dries up, and still gives the same re-actions with iodine.
4. Passes into a brown fluid on the addition of sulphuric acid <i>concentrated</i> .	4. Swells in, but does not dissolve with sulphuric acid, with change of colour.
5. Soluble in ether.	5. <i>Not</i> soluble in ether.

By way of chemical analysis very trustworthy results seem to have been arrived at by Friedreich and Kekulé. On submitting the white amyloid matter to ultimate analysis they obtained the following composition in equivalents per cent. (*Med.-Chir. Review*, 1861, p. 59).

	C.	H.	N.
<i>Amyloid</i> , ...	= 53·58	7·0	15·04

Now, the composition of albumen, according to Dumas and Cahours, Lieberkühn and Rüling, is as follows:—

	C.	H.	N.
<i>Albumen</i> , ...	= 53·5	7·1	15·8
Dumas and Cahours, {	53·4	7·2	15·7
	53·5	7·3	15·7
Lieberkühn, ...	53·5	7·0	15·6
Rüling, ...	53·8	7·1	15·5

Surely these results show an almost perfect chemical identity between albumen and the morbid substance found in the so-called waxy spleen; and demonstrate that the waxy degeneration, in the spleen at least, is due to a peculiarly modified albuminous material, and not to starch? On the other hand the chemistry of the corpuscular variety of the *corpora amylacea* occurring as a deposit in various parts—*e. g.*, in the brain, the prostate, and the *ependyma* of the ventricles—shows a re-action almost identi-

cal with starch. The corpuscles also have concentric laminæ, and, according to some, resembles starch-granules when polarized. As regards the corpuscles of the prostate, sugar has been chemically produced from them, and demonstrated by Trommer's test.

Many of these corpuscular varieties of amylaceous bodies are no doubt of the same nature as starch; and therefore the direction which inquiry ought now to take will be to determine "Whether or not there is any chemical affinity on the part of the formless matter of waxy degenerations with the corpuscular variety of the amylaceous concretions?" Such an affinity has been assumed hitherto; but, so far as observation has gone, the evidence of any affinity seems to be getting less and less. On the other hand, the modifying effects of admixture and of growth are very remarkable as regards these prostatic concretions. *Some* of them *iodine* will not colour blue, not even after sulphuric acid has been added; and as growth proceeds, any amyloid matter they contain gradually disappears. Many admixtures of organic and inorganic substances give various shades of colour; and the yellow-brown coloured deposits failed to give forth sugar to Paulizky's attempts.

General Characters and Anatomical Description of Tissues which have undergone Amyloid Degeneration.—The cut surface of an organ so affected has a semi-transparent appearance. It feels like a piece of soft wax, or of wax and lard combined (WILKS). It cuts into portions of the most regular shape, with sharp angles and smooth surfaces; and the thinnest possible slices may be removed by a sharp knife for microscopical examination without any special preparation. The tissue is abnormally translucent. Water and alcohol, acids and alkalies do not produce any change upon the transformed parts, which may be kept for a length of time without decomposition. The organs affected are increased in volume, in solidity, and in weight, absolute and specific. Anæmia is predominant; but the colour of blood or of tissue shines through the semi-transparent morbid substance.

Amyloid degeneration is generally widely diffused; so much so that a constitutional state of ill-health seems associated with its production; and in cases preceded by a local disease, such as caries of a bone, the degeneration may be found in the adjacent lymphatic glands only (BILLROTH). This is the earliest appearance of the degeneration yet recognized.

The small vessels of the tissue—the more minute arteries in

particular—are, as a rule, the first structures attacked. The coats of the arteries become thickened and granular, and at last pellucid, transparent, and hyaline. Their calibre is reduced, and their cut section remains patulous.

It is the middle or muscular coat of the vessels which first changes. Each fibre-cell becomes a compact hyaline, pellucid, transparent particle, with an indistinct outline, and all the tissue involved becomes at last uniform, clear, and transparent. The degenerate artery looks like a compact, homogeneous, silvery cord or thread, of a clear and glassy appearance, with a lustre like molten glass without polish, or like rough ice. This colourless, hyaline, degenerate tissue is very tough, but not hard nor brittle, like the calcareous degenerate parts. All other degenerations tend to obscure the original texture, by making it more opaque. This degeneration, on the contrary, renders the affected tissue more transparent and pellucid. It is in reality a *hyaline degeneration*. The specific cells of the functional parenchyma, when the degeneration affects a solid organ like the liver or kidney, next undergo the change, which finally spreads to the nutrient vessels amongst the connective tissue.

When a solution of iodine is brought in contact with the affected part, a very deep violet-red colour is produced; and this deep-red colour seems to be *alone* a sufficiently characteristic test of the existence of the degeneration, especially when in a few seconds the colour increases in depth from the moment it takes effect. It is a re-action which ensues between the iodine solution and the morbidly degenerate part. The best test-solution is composed as follows:—*Twelve grains of Iodine* is to be dissolved with *twenty-four grains of Iodide of Potassium*, and mixed with *three ounces of Water*. Such a test-solution ought always to be at hand in the dead-house, or on making a *post-mortem* examination anywhere.

Elements of Tissue in which Amyloid Degeneration has been demonstrated.

1. Nervous System:—ligamentum spinale cochleæ: atrophied parts of brain and spinal cord: gelatinous softening, and tumors.

2. Spleen:—cells of the Malpighian sacculi and pulp: thickened walls of the arteries in all stages: the trabeculæ.

3. Liver:—the hepatic cells and intralobular vessels (hepatic arteries): intercellular tissue.

4. Kidneys:—Malpighian tufts and afferent vessels, the walls

of which become enormously thickened: areolar tissue in the vicinity of the papillary ducts.

5. Muscular tissue of the heart and the uterus.

6. Blood-vessels of the villi and mucous membrane of the alimentary canal.

7. Osseous tissue.

8. Lymphatic glands.

9. Besides the original structures of the body, old deposits in serous membranes, having lost their fibrous character, becoming dense, more vascular, and semi-transparent, undergo this metamorphosis (GAIRDNER).

10. Tubercle also becomes amyloid (GAIRDNER).

11. The cancerous nodules in a waxy liver also become amyloid (GAIRDNER).

12. In some cases of inflammation with exudation on the mucous membrane the exudation has assumed the amyloid degeneration (VIRCHOW).

13. The fibrine of a hæmatocele (FRIEDREICH).

The extensive range of organs in which this remarkable degeneration has now been demonstrated clearly shows that the lesion cannot be regarded as merely of local importance. Its occurrence seems rather to point to some general pathological state of which the degeneration is the expression. In the first instance it is found more particularly affecting the functional capillaries of the most important organs of the body—*e. g.*, the kidney, the liver, the spleen, the intestines, as well as the minute arteries of nutrition of those organs, and of the pia mater, bone, and lymphatic glands. The results of such a degeneration must therefore be sooner or later destructive,—(1.) To the function of the invaded organ; (2.) To its nutrition; and we can only arrive at a correct pathology of this degeneration by a close observation of the circumstances, condition, relations, and symptoms under which the lesion manifests itself. These must be studied especially in relation to the functional or physiological anatomy of the organs implicated. As yet the lesion has been recognized with certainty only in the dead-house. There it has been found associated with certain diseased states; and all the cases agree in this particular, namely, that the constitution of the patients has been broken up by ill-health (*cachexia*) of some considerable duration before death. So it has been amongst the soldiers dissected at Fort Pitt and at Netley; and the following statement

is a summary of diseased conditions, in the order which furnishes the greatest number of cases of amyloid:—

Diseased States with which Amyloid Degeneration has been found associated, or upon which it is engrafted,—

1. *Diseases of the bones*, especially caries and *necrosis* in scrofulous subjects. *Rickets* also leads to the amyloid liver and spleen, as observed by Glisson, Portal, Rokitansky, Lambe, Loeschner, Frerichs, and Jenner.

2. *Syphilis*, especially in its ulcerated forms, the cachexia having been prolonged. Syphilitic children have been the subject of it when newly born.

3. *The malarious cachexia*, especially *intermittent fever*.

4. *Mercurial cachexia* and *marasmus*.

5. Pulmonary and intestinal forms of *tubercle*.

6. *Albuminuria* and *anasarca*.

7. *Diseases of large arteries*.

As to the origin of the lesion or degeneration, Frerichs has propounded two questions, namely,—(1.) Is the lesion due to deposits from the blood of the amyloid, waxy, or albuminoid matter in some primordial form, and which is generated in the blood, in consequence of a local disease, such as caries of the bones? (2.) Is the amyloid or waxy matter developed locally in the affected tissue by the transformation or degeneration of the tissue into albuminous matter?

It has been more recently urged by Dr. Dickenson that the degeneration is always the result of extensive purulent formation of long duration.

Arguments are put forward by Virchow and Frerichs to show that the lesion may be due to a deposit from the blood; but the weight of evidence seems, on the whole, to point to a peculiar degeneration of existing tissue. For, (1.) In cases where the lesion follows affections of the bones, the lymphatic glands adjoining the diseased bones are implicated before the kidneys, liver, or mucous membrane of the intestines. (2.) General causes of ill-health (cachexia), pointing to impoverished blood, are in operation, and organs situated in different parts of the body are simultaneously affected. (3.) The fibrin of the blood itself has been observed to undergo the degeneration; for Friedreich found a substance which gave the amyloid re-action with iodine in the old fibrinous layer of the sac of a hæmatocele.

In this remarkable degeneration an acquaintance with a new fact in pathology must be recognized—*i. e.*, since 1854—associating itself with grave constitutional disease, and distinguished from every other morbid condition hitherto known, by the physical, chemical, and physiological characters just described.

The Clinical History of amyloid degeneration is remarkably deficient. The effect of the degeneration is to interfere with function of organs and nutrition of parts; and the injurious effects are the more marked as the lesion extends through many important organs. For example, hepatic cells cease to take part in the formation of sugar or the secretion of bile. Blood-vessels lose their power of transmitting fluid through their walls, and become impervious as to their canals. Hence those who suffer from amyloid disease have an appearance of *general ill-health*, denoted by paleness of the surface, by symptoms of anæmia, hydræmia, or by leukæmic affections of the blood; and the more so as the constitution is enfeebled by such morbid processes as ulceration of bones, syphilis, tuberculosis, or malaria. The sequence in which the different organs degenerate is uncertain. In most cases of caries and necrosis the kidneys seem to be first attacked after the lymphatic glands. In cases of intermittent fever it is usually the spleen which is first affected; and generally it seems rare to find several, or all the organs affected to the same extent.

Signs or Symptoms associated with this degeneration discoverable during life.—On these points data are wanting upon which to found any statement. The pathological change is of so recent discovery that well-recorded cases, terminating in death, with verification of the symptoms by *post-mortem* inspections, are very few indeed. There is no subject, therefore, more full of interest, or one more likely to repay close observation and well-directed pathological inquiry, than the diagnosis of amyloid degeneration. Cases in hospital ought to be most carefully noted, and especially such ambiguous cases as those where marasmus, anæmia, or dropsy are *primary* symptoms, and which are not to be accounted for even after the blood has been examined microscopically during life, and the condition of the liver, heart, spleen, and lymph-glands carefully inquired into, without evincing signs of disease. In a remarkable case recorded by Friedreich and Kekulé, and quoted in the *Medico-Chirurgical Review* for October, 1860, *diarrhœa* and *vomiting* were of frequent occurrence, with a systolic murmur of the

heart, and high-coloured and albuminous urine, with a specific gravity of 1·019. The patient, a female, after suffering from tertian ague for twelve months, became dropsical and emaciated. The intestines throughout, the stomach, the colon, the jejunum, and especially the capillary vessels of its villi, were affected, as well as the vessels of the kidneys. The urine should be watched as to albumen, or deposits, and its amount in relation to body-weight should be recorded. When albumen appears, it goes on gradually increasing; and hyaline casts increase with the increase of albumen.

Dr. Stewart, of Edinburgh, records twenty cases, *and nine dissections* in cases of Bright's disease, where he considered amyloid degeneration to have been present (*Edinburgh Medical Journal* for February, 1861). He records that large quantities of urine were passed in the early stage, of supposed waxy degeneration, and of a specific gravity from 1·005 to 1·015. In all the cases there was a striking general correspondence in the other symptoms; and Dr. Stewart thinks that from this similarity of symptoms, and from other considerations, he is warranted in believing that amyloid degeneration existed in the *eleven* cases that did not die. The history of all Dr. Stewart's cases is markedly different from that of the fatty kidney which Dr. Bright figured in his first plate, and illustrated in his first case. Almost all of the cases were associated with long-continued wasting disease; and it has been long known that the form of renal affection accompanying phthisis, syphilis, and other wasting maladies, is the waxy or amyloid degeneration. Of the twenty cases related by Dr. Stewart, six were associated with phthisis, six with syphilis, two with caries, two with intemperance, one with cancer, one with chronic rheumatism, and two with no particular disease.

The degeneration is much more common than is generally supposed. It has been observed very frequently amongst the soldiers who have been dissected at the Military Hospital for invalids, formerly at Fort Pitt and now at Netley. The microscope and iodine test can alone determine its absence; and without microscopic examination the absence of the degeneration cannot be determined. For a detailed account of "amyloid degeneration" in the various organs, see the description given under LOCAL DISEASES.

Before stating the principles which dictate the treatment of

the complex morbid processes just described, and of individual diseases in particular, it behoves the student, first, to make a separate study of *the varying types of diseases, their prevailing peculiarities, and the constitutional tendency to change of type which they assume* at varying intervals of time; and, second, to observe and learn to recognize *the various modes by which diseases terminate fatally*.

CHAPTER IX.

TYPES OF DISEASE AND THEIR TENDENCY TO CHANGE.

IN describing, appreciating, or ascertaining the type of a disease, our attention must be directed to a variety of phenomena and conditions; and the type of the disease only becomes characteristic and distinctive when some one or other of those conditions becomes predominant, or manifests itself more decidedly than others. The hereditary or natural constitution of the individual may be regarded as an important element in determining the type of the disease. Town life, as compared with country life, also exercises an influence on the type of many diseases; and there are good grounds for believing that the town life and artificial habits of the present period are more prejudicial to the strength of the constitution than those which prevailed when large towns were but rural villages, and the inhabitants more simple in their mode of life, and less artificial in their habits.

The occupation of the individual in many instances exercises an influence over the complex processes of disease; and there cannot be a doubt that some diseases have altogether disappeared, while others have been so much modified that their resemblance to the original form or type can with difficulty be recognized.

With regard to Edinburgh and its vicinity, Professor W. T. Gairdner observes that the *changes of type* which have occurred in epidemic fever, and especially in typhus fever, during the ten years previous to 1862, or since the cessation of the great epidemic of 1847-8, are not less remarkable than the diminution in the amount of this class of cases. The *relapsing fever*, or *synocha*, which formed so large a part of the epidemics of 1843-4 and 1847-8, has for the time absolutely disappeared. *Typhus*

fever has become less fatal to those attacked than it was ten years ago; while its general type, and some of its leading characters have been remarkably modified. This is especially noticeable in the *diminished mortality*; the *earliness of the appearance of the eruption*; the *earliness of the crisis* (tenth to fourteenth day as a rule, and rarely prolonged into the third week)—a great cause of the diminished mortality; for a day's delay of the crisis, in a case of any degree of severity, is an immense addition to the risk, (*Clinical Medicine*, 1862, p. 156, *et seq.*) Nevertheless, it is the rule that diseases preserve their essential characters and nature from age to age, although the opinions of the profession respecting them and their treatment may change from year to year, or from one period of time to another. For example, *small-pox*, *measles*, *typhus fever*, *typhoid* or *intestinal fever*, *dysentery*, *diphtheria*, and the like, always remain the same as to their essential characters, and unchanged in their special symptoms; but it must be remembered that they are also very often modified in their phenomena by the existence of such constitutional ill-health (cachexia) as may arise from *syphilis*, *mineral*, *vegetable*, or *alcoholic poisoning* of the system, as well as from *gout*, *rheumatism*, *scrofula*, *tuberculosis*, and *scorbutus*, from epidemic causes; and especially the artificial mode of life in towns as compared with the more natural habits of rural villages. These are the diseases and the conditions of existence which are the main source of the deterioration of the human race, in all physical attributes, among such civilized communities as our own. Diseases also have arisen which appear to be more or less new to us, in some instances resulting from a hybrid combination of various pathological phenomena to be noticed presently. While this is undoubtedly the case, there is abundant evidence to prove that we now have in some respects a more healthful enjoyment of our life on the whole (although the constitution may not be so strong) than formerly; and that the duration of man's life of late years has been, on the whole, prolonged. While it is the lot of "all men once to die," that final change is more frequently deferred than was wont, to beyond that period when, in the words of the inspired Psalmist, it is recorded that "the days of our years are threescore years and ten; and if by reason of strength they be fourscore years, yet is their strength labour and sorrow."

It has been observed by a popular writer that there never

were any specifics discovered against the *plague*, the *sweating sickness*, or the *leprosy*, and yet these diseases, so far as regards this country, are now amongst the things that were, and are almost unknown. They have disappeared, not before any marvels of medicine, or any perfection of chemical sciences, but as a consequence of the gradual amelioration of our condition through sanitary improvements. Observe, also, what sanitary science has done, in a comparatively short space of time, to ameliorate the condition of the British army. The Right Honourable the Secretary of State for War (Sir George C. Lewis, Bart.), in moving the army estimates for the year 1862 in the House of Commons, said,—

“Improvements have been introduced with a view to ameliorate the social, moral, and sanitary condition of the private soldier. Much expenditure has been incurred for the sake of enlarging and improving barracks, and in carrying out various recommendations of the House of Commons with respect to barracks and the hospitals connected with them. I am happy to say,” continues the Right Hon. gentleman, “that these efforts have not been unattended with important results, as will appear from authentic returns of the mortality in the service. These returns have been prepared by the Director-General of the Army Medical Department, and I believe they are perfectly authentic, though it is certainly difficult to believe that so great a change can have taken place in so limited a period. It is possible that the greater youth of some portions of the army may, to a certain extent, affect the returns, but I believe the difference is mainly to be explained by improvements in the sanitary conditions under which they are now called on to serve.

“DEATHS AMONG THE TROOPS SERVING IN THE UNITED KINGDOM ANNUALLY
PER 1,000 OF MEN.

	From 1830 to 1836.	1859 to 1860.
Generally throughout, ...	14	5
Cavalry of the Line, ...	15	6
Royal Artillery, ...	15	7
Foot Guards, ...	21	9
	From 1836 to 1846.	1859 to 1860.
Infantry of the Line, ...	17	8

“SIMILAR RETURNS FOR THE COLONIES ARE AS FOLLOWS:—

	From 1837 to 1856.	1859 to 1861.
Gibraltar, ...	22	9
Malta, ...	18	14
Ionian Islands, ...	27	9
Bermuda, ...	35	11
Canada, ...	20	10
Jamaica, ...	128	17
Ceylon, ...	74	27

"I have other returns from other colonies," continues the Right Honourable gentleman, "and I believe that these returns are authentic; and certainly they show how very considerable a diminution has taken place in the mortality of the army" (*Times*, March 4, 1862).

The late Lord Herbert was the main agent in accomplishing this great work, which, as years pass on, will become better appreciated, more widely known, and more energetically followed up.

Professor W. T. Gairdner has happily observed, that in proportion as we are getting rid of the severer forms of epidemic disease (*e. g.*, fever, dysentery, scurvy, influenza, all more or less preventible), which had deteriorated the health of the population previously to 1848, we are also getting rid of the more severe and unmanageable types of acute inflammation. Inflammatory diseases, like fevers, he therefore justly considers to be subject to epidemic causes of increase and diminution, both as regards frequency and severity; and he believes that the acute inflammations are nearly as much under the influence of the sanitary reformer as the more obviously epidemic fevers; and, further, that some even of the chronic organic diseases have already yielded, and may be expected still further to yield, to the improved habits, the better clothing, the greater abundance of food, and the diminished destitution of the population generally (*Clinical Medicine*, p. 42).

The Art of Medicine, guided by Sanitary Science, must now, therefore, be regarded as a productive art; for by diminishing the occurrence of preventible disease, and thereby lessening mortality, the average duration of human life has been extended to an age nearer that which has been ordained for man. Nevertheless, it behoves the Physician to remember that the sphere of his professional exertions is limited, and surrounded by insurmountable barriers;—that death will eventually come alike to all, "reminding us that we ourselves must become victims to the incompetency of our art."

Dr. Pollitzer, of the Children's Hospital at Vienna, has expressed the opinion, that while the duration of mortality at early ages is diminishing in all civilized countries, under the various influences of extended hospital accommodation, care of the sick, vaccination, and general sanitary regulations, there is no corresponding increase in the strength and vigour of the human race. On the contrary, the boundaries between health and

disease are becoming less and less marked. There are now to be observed numerous conditions which are undoubted deviations from the healthy standard, which it is impossible to delineate or accurately to define, because they make their appearance during a state of "*relative health*." The physician is not always even able to name the disease; and while the patient maintains that he is not feeling in health, and not looking in health, but wasting away, his food doing him no good, he has no alternative but to call himself ill. Such is the insidious mode in which many of those truly CONSTITUTIONAL diseases and DEGENERATIONS make their appearance, and which may be regarded as constituting a peculiar characteristic in the diseases of our times. These diseases are known by the various names of *anæmia*, *spanæmia*, *leucocythæmia*, *chlorosis*, to which we must also add such degenerations as those described in the previous pages, namely, "amyloid degeneration," "pigmentation," "fatty degeneration," and the ill-health (*cachexia*) of malaria, and of syphilis.

The poorness of the blood, peculiar to the class of diseases mentioned, furnishes the soil in which the feebleness and deterioration of our race is most unmistakably evident. The nervous system is, moreover, extensively involved in the diseases of the age, and thus feebleness and debility constitute their dominant character. This physical deterioration is held to be a "*sad memorial of modern civilization*." In this respect the observations of Dr. Pollitzer coincide with those of Dr. Forbes Winslow. "A constant stretch of the mental powers, a restless excitement of the passions, a perpetual struggle for advancement, the fresh wants of every day (science, and the arts themselves, being subservient even to the luxury and demoralization of the times), the destruction of all moral harmony and peace, are evils which undoubtedly prevail, and which re-act especially upon the younger generation." The sins of the fathers are thus being visited upon the children. The demands made upon the youth of eighteen or twenty of the present day would formerly have been considered a sufficient tax for the strength of a man of upwards of five-and-twenty. Many of the features, also, which characterize the pathology of our age have arisen out of the treatment of infancy and childhood; and much of the deterioration of the race at large may be shown to date its origin from childhood. Thus, after seventeen years' observation in children's disorders, Dr.

Pollitzer writes that *anæmia* and *chlorosis* occur alone or associated with *rickets*, *hypertrophy* of the *lymphatic glands*, and of the *spleen* and *liver*, to an incredible extent even from the first month of life. In the children's hospital at Vienna, from seventy to eighty *per cent.* he found to be thus affected. Wherever the nutrition of the child had been imperfect, the constitutional diseases associated with poverty of the blood became widely diffused. The stomach and intestinal tract first suffer, constituting the prevailing morbid condition of childhood—materially influencing the mortality at an early age, and if the age of childhood is survived, affecting the future health of youth and manhood, and doubtless of subsequent generations (*Med.-Chir. Review*, Report on Medicine, p. 261, July, 1857).

The types of disease are also evidently modified by complication with other diseases, now more widely spread; and the doctrine of the incompatibility of two or more contagious diseases concurring in the same subject has been clearly proved to be erroneous. Dr. Murchison, in an admirable paper on this subject in the *British and Foreign Medico-Chirurgical Review* for July, 1859, has clearly shown the co-existence of *variola* and *scarlatina*; also, of *variola* and *rubeola*. Dr. F. J. Brown, of Rochester, has recorded a case of *variola* concurring with *measles* and *purpura*. The co-existence of *variola* and *roseola* or *erysipelas*, of *variola* and *pertussis*, of *variola* and *varicella*, of *variola* and *vaccinia*, of *vaccinia* and *scarlet fever*, of *vaccinia* and *rubeola*, of *vaccinia* and *pertussis*, of *vaccinia* and *varicella*, of *rubeola* and *pertussis*, of *variola*, *rubeola*, and *pertussis*, of *scarlatina* and *rubeola*—the *rötheln* of the Germans—of *scarlatina* and *enteric* or *intestinal fever*, of *typhus* and *enteric*, *intestinal*, or *typhoid fever*, have all been more or less clearly shown. Virchow relates a case of typhoid fever, combined with striking symptoms of cholera, occurring at Würzburg. Typhus fever and the marsh fevers have been observed to occur together (PRINGLE). Bilious remittents have prevailed with small-pox in the West Indies, forming, as an old writer remarks, “the most infernal combination that ever affected the human frame.” Yellow fever has been found associated with putrid typhus; while specific yellow fever and marsh fever, with phenomena similar in many respects to specific yellow fever, undoubtedly occur together.

There are good grounds for believing that as we approach

certain well-marked geographical regions of the earth, where characteristic types of disease prevail, the confines of these disease-realms are found to mingle their types of disease together, so that the diseases of one region merge into and participate in many of the characters peculiar to the other. Cholera has extended its ravages over the earth, and is now a disease whose germs are endemic in our land; and the system under its influence, especially in the early cases of an epidemic, becomes rapidly depressed, to the speedy extinction of life. The *furunculoid* epidemic which prevailed about eight years ago was one of a novel variety. The *black death* of the fourteenth century seems to have revived in India, and is described by the name of the *Indian Pali plague*; and it may be that the formidable disease which laid waste our country in the thirteenth century may have arisen in these districts, and proceeded thence to our land, passing apparently in the same way that cholera has done (DR. ALLEN WEBB).

Our modern treatises on medicine justly and properly deal, largely and minutely, with the descriptions of individual diseases, as far as their nature can be discerned; and the languages of all civilized nations attempt to define and describe them clearly. Now only are we beginning to profit by an extended inquiry into the diseases of nations; and to find that as man wanders from his native home, the type of the diseases to which he is liable also changes. In this field of science an unmeasurable and still unexplored country extends on every side. The more minutely, also, that individual diseases can be defined and described, the more useful will such descriptions be for comparison in future ages; and it will be seen, on comparing the descriptions of diseases in times past with accurate descriptions of the same diseases now, how, at various periods and under various circumstances, the expression of certain sets of symptoms becomes sometimes strongly developed, while at other times the same classes of symptoms were mild and subdued; but the really essential characters of specific diseases and morbid processes remain as persistent as the specific characters of a plant or an animal.

It will be seen how, in epidemics, many diseases have been characterized by the expression of malignant phenomena scarcely perceptible before. The small-pox now-a-days is not the malignant small-pox of the time of Sydenham. Nevertheless, it is

small-pox. And although it may be said that such an example does not illustrate a change in the type of a disease, because the change has been effected by artificial means; yet it must appear evident that, in effecting this change, natural results have only been imitated; and who can tell what modifying influences of a similar kind are going on, although the science of medicine can as yet take no cognizance of them? We know that certain diseases confer immunity on the individual from future attacks; may it not be, therefore, that immunity to individuals from some diseases is conferred by agents and processes of which we as yet know nothing; and that ultimately the types of complex morbid states may still come to be very much changed from what they are now? They appear to be changed, according to the best authorities, from what they were fifty years ago. "Many of the symptoms, and particularly the constitutional fever usually attending internal inflammations, and resulting from cold, or from other causes independent of the application of morbid poisons, are liable to variation in like manner, although not so decidedly nor so rapidly as the epidemic diseases, in the course of time, and from causes not yet known. They have in fact undergone very considerable change since the early part of the present century; and it is on this account that inflammations of the lungs in particular are treated with equal success at present, with a much smaller loss of blood than they used to demand" (ALISON).

Such changes in the types of disease were formerly observed and much insisted upon by Sydenham, especially in the progress and recurrence of continued fevers; and it is now a fact well recognized, that not only does the prevalent mode of fatal termination during epidemic diseases vary, but so also do the types, peculiarities, and morbid constitutional tendencies vary in these diseases. It is chiefly with regard to the *local*, *sporadic*, or *intrinsic diseases*, and especially inflammations, such as the *cephalic*, the *pulmonic*, or the *enteric*, that any doubt exists as to whether or not they vary in their type. Distinct statements as to this fact, however, have now been made by many accurate observers, whose experience is of the utmost value to science. Dr. Alison and Dr. Bennett both agree as to the fact, "that of late years, and apparently also in different parts of the world, *inflammation*, the most important of all forms of *local diseases*,

seldom shows itself with such general symptoms as demand or would justify, in the opinion of the practitioners treating them, or indeed could bear, the large bleedings which were formerly regarded as the appropriate remedy, and which accordingly are now seldom practised." There are not only also fewer examples of violent inflammation of the lungs to be met with, but the *usual* (highly inflammatory) type of fever attending such inflammation has materially changed, as occurring in the present day. This change which has taken place in the *type* of the usual phenomena characteristic of inflammatory fever, cannot be explained merely by the circumstance that a previously enfeebled or diseased state of the system has brought it about in the individual. The inflammations of internal parts, such as pneumonia, now *occur often without febrile re-action*, and neither demanding nor bearing full bleedings, as described by Cullen and other authors. It is consistent, moreover, with the extensive experience of Drs. Alison, Christison, Watson, and many other physicians of the greatest eminence and long experience, that the inflammations now seldom occur with such severe symptoms of inflammatory fever as have been described at page 104, and which were the rule in the time of Cullen and of Gregory. The constitutional symptoms now attending such inflammations partake more of the type of the so-called *typhoid* state, and independent of any epidemic influence or poison having acted on the body.

The constitutional symptoms for many years past accompanying pneumonia, for instance, in this country, have been of the following kind:—An enfeebled circulation; softness of the pulse, and easiness of depression by depletion, or even by taking the erect posture; tremors and feebleness of voluntary muscular motion, approaching to subsultus; indifference to surrounding objects; sickness and vomiting in some cases, with dryness of tongue and lips in others; complete anorexia or depraved appetite; in all, the symptoms generally tend to assume those of the so-called *typhoid* state rather than of *inflammatory fever*. Nevertheless, in the treatment of such cases, blood taken *sufficiently early* is distinctly beneficial; and although it may show the *buffy coat*, this has neither the thickness nor the tenacity of the *fine buffy coat* seen and described in former days; faintness supervenes on the loss of a quantity small in comparison of what was formerly well borne, and there is no

such encouragement to a repetition of the bleeding, from the pulse speedily regaining its strength, or from the local symptoms abating and quickly recurring, as was formerly noticed by Cullen, Gregory, Christison, Alison, and other veterans in the practice of medicine. Dr. Gairdner also points out the great distinction to be drawn between the doctrines of Alison and of Todd in this respect. Dr. Alison recognized the disuse of blood-letting, and the increased necessity for stimulants, as a *consequence* of the changes he observed in the character of diseases. Dr. Todd, on the other hand, disowned entirely the idea of such a change, and came to regard the administration of stimulants as a matter of routine. The former recognized the altered type of disease, the changes observed in its physiological manifestations, and the gradual disappearance of those forms of acute inflammation which before had appeared to require and to bear blood-letting. Dr. Todd did not recognize or acknowledge these changes (*Clinical Medicine*, 1862, p. 34).

The mode of fatal termination is also different. The tendency to death is most usually either by *coma* or by *asthenia*, as in typhus fever. The tendency to gangrene is said to have become more frequent in various inflammations of internal parts, and particularly in the lungs, within the last fifty years, than it was in the time of Cullen and of Gregory. Such are the records of experience, as stated by the late Dr. Alison in a series of papers published in *The Edinburgh Medical Journal* in 1855, 1856, and 1857; than whom the opinion of no one is more entitled to respect and consideration. "Although dead, he yet speaketh."

Dr. Handfield Jones, physician to St. Mary's Hospital, London, observes, "that instances of impaired action of the heart, sometimes amounting to serious danger, are met with at the present time with an absence of all conditions which require or justify venesection. This is associated with other symptoms of depressed nervous power, which appear to be most reasonably attributed to some kind of epidemic influence much resembling that of malarious diseases.

The type of disease which reigns at present is believed by some to have prevailed more or less since the appearance of pestilential cholera amongst us. There is now little of sthenic inflammation; bleeding and tartar emetic are but sparingly

needed, while quinine, strychnine, and other tonics, with opium, and cod-liver oil, are continually in requisition in our efforts to raise and maintain failing power (*Medical Journal*, March, 1857). According to the experience of obstetric practice, the observations of Mr. Sidey, of Edinburgh, are to the same effect. He records that the epidemic of influenza in this country upwards of thirty years ago was of a most inflammatory character; cases of influenza now generally assume the *asthenic* type, and demand a different treatment. Moreover, he observes that it is consistent with the experience of the veterinarians that the change in the type of disease has been observed among the lower animals to the same extent as in man.

In 1782, when Dr. Hamilton published his memoirs on the "*contagious catarrh*," or *influenza*, he records distinctly his belief that "our constitutions are considerably changed within the last century in Great Britain. Diseases," he remarks, "in their nature phlogistic (*e. g.*, *measles*), have appeared within the thirty years previous to 1782 less inflammatory than they formerly were, and accompanied with a considerable degree of putridity;" and blood-letting in the "*contagious catarrh*," he states with emphasis, cannot be tolerated. There seems, therefore, to have been even then an increasing belief that the degeneracy of the human race, as a whole, is in some respects advancing; and there seems some visible evidence of this more or less traceable through the past four generations.

Morel especially directs attention to the apparent increase in Europe of mental alienation, and of those abnormal states of existence which have special relations with the occurrence and existence of physical and moral degeneracy in the world; and if a comparative increase in the number of the insane cannot yet be proven, there would seem to be a tendency to more frequent complications among them of those morbid states which diminish the probability of cure, such as *general paralysis*, *epilepsy*, and a marked depression of all the intellectual and physical forces, which depression is consistent with the *asthenic* phase of present existence. *Hysteria* and *hypochondriasis*, formerly almost the exclusive appanage of the rich, the indolent, and those of a wasted life, are known to attack in great proportions the working and the agricultural classes, among whom suicidal tendencies prevail.

Dr. Forbes Winslow writes, with regard to nervous diseases,

that cases of disease of the brain and nervous system are now not only of more frequent occurrence, but that a certain unfavourable type of cerebral disorder develops itself in the present age at a much earlier age than formerly. Softening of the brain, for instance, often manifests itself at the early age of thirty and thirty-five. The brain in the present day is over-worked, its psychical functions are unduly exercised, strained, and taxed in the great effort required in the severe struggle and battle of life to obtain intellectual supremacy, professional emolument, and status (*Journal of Psychological Medicine*, July, 1857).

Morel again shows how imbecility, congenital or early acquired idiocy, and other more or less complete arrests of development of the body, and of the intellectual faculties, indicate the existence of children who have acquired the elements of their degeneracy during intra-uterine life. It behoves, then, all civilized governments anxiously to inquire into and to consider such facts as show,—(1.) The continued increase of suicide; (2.) The continued increase of crimes against order and law, or against the person; (3.) The monstrous precocity of young criminals; (4.) The abnormal conformations of the skull, and tendency to early union of the cranial sutures, which prevail among criminals. So much is this the case, that in large towns, or in penal settlements and convict prisons, the physiognomy and general aspect of the “criminal population” are characteristic of a class; (5.) The general diminution of the intellectual powers, with the manifestation of the most depraved immoral tendencies; (6.) The increase in the inmates of asylums and prisons; (7.) The etiolation, blanching, anæmic or cachectic condition of the population, and any relative increase of such constitutional diseases as *anæmia*, *chlorosis*, *gout*, *Bright's disease*, *rheumatism*, *melituria*; (8.) The increased development of paralytic and convulsive affections. These are the main directions in which the degeneracy of the human race is demonstrable; so much so that in some localities the inhabitants can no longer fulfil the conditions required for military service (*Traité des Dégénérescences Physiques, Intellectuelles et Morales de l'Espèce Humaine, et des Causes qui produisent ces Variétés Maladives*. Par le Dr. B. A. Morel, de l'Asile des Aliénés de S'Yon, 1857).

The principal sources of degeneracy which appear at present

to be most active in their influence for evil on large masses of mankind may be stated as follows:—(1.) Degeneracy from *Toxæmia*, as from the abuse of alcoholic fluids, opium, preparations of Indian hemp (hachisch), tobacco, and the like; also from the effects of mineral poisons, such as lead, mercury, arsenic, phosphorus; and from the use of unwholesome vegetable food, such as diseased rye, maize, wheat, and the like. (2.) Degeneracy from the persistent and pernicious influence of *malaria*. (3.) Degeneracy from certain peculiar geological formations, soil, and water, as in the development of goitre (MACLELLAN, WATSON). See Paper on Hygiène of India, in *Med.-Chir. Review*. (4.) Degeneracy from the effects of epidemic diseases which now and then afflict large populations, profoundly influencing the system, and engendering those morbid temperaments whose types are more fully expressed in the generations which follow the one that has suffered from such epidemic pestilences. Many of such-like epidemics act like toxic agents on the nervous system. (5.) Degeneracy from the effects of the “*great town system*,” as the phrase is. The chief elements of such degeneracy are, (a.) unhealthy situations, (b.) a noxious local and general atmosphere, (c.) insufficient air, (d.) insufficient and improper nourishment, (e.) deleterious avocations, (f.) moral and social misery, wretchedness, and crime. (6.) Degeneracy from fundamental morbid states, congenital or acquired, as seen in the imperfect cerebral developments, deaf mutism, blindness, constitutional diseases and diathesis (implanted, hereditary, or acquired), such as syphilis and scrofulosis. (7.) Degeneracy from mixed causes, from marrying in and in; and from other causes not included in the above (*Med.-Chir. Review*, Jan., 1858).

By far the most active sources of degeneracy are thus seen to be those direct and repeated influences upon the blood, the brain, and the nervous system which give rise to special morbid conditions; and which often place those who periodically expose themselves to the influence of toxic agents in a condition verging on or equivalent to insanity. The effects of *chronic alcoholism*, for instance, in giving rise surely and progressively to the *degeneration* of the individual, are mainly demonstrable by the induction of the following states, namely:—persistent loss of appetite, indigestion, nausea; occasional diarrhoea, progressive emaciation, and cachexia; the appearance of pustular eruptions;

the occurrence of eructations associated with offensive breath; serious disturbance of the function of the stomach, liver, kidneys, and heart, and the production of organic lesions in these organs, and in the structures of the blood-vessels, followed by fatal *serous effusions, dropsy, hæmorrhages, extravasations, or apoplexies*. Intercurrent with these morbid conditions, at variable periods, and otherwise contingently, "fits of drunkenness," with sexual incompetency, different forms of psychical aberration, *delirium tremens*, suicidal melancholy, and such-like morbid phenomena ensue. Finally, *epileptiform seizures, general paralysis, drivelling or slaving idiocy*, may close the scene. Those who become thus degenerate by alcoholic poison are arranged by Morel into two classes, namely,—*First, Individuals who arrive at length, by a series of well-marked nervous lesions—physical and intellectual—at general paralysis. Second, Individuals who, although profoundly affected as regards enervation, still remain stationary at a certain state, leading a miserable existence, characterized physically by a special condition of ill-health (cachexia and marasmus), and morally by a manifestation of the worst tendencies, and of the lowest animal propensities.* The serious degenerative effects thus detailed in their extreme forms, resulting from the poison of alcohol, ultimately influence the procreative functions; first, by diminishing the vital standard of the offspring; and second, by annihilating the generative power altogether. When such results are coupled with the moral and social aberrations which ensue from bad example, misery, and want, in families and masses of men, they become ample sources of the degeneracy and degradation of the population, not only throughout the existing but succeeding generation; and not only is the vice of alcoholic abuse hereditarily transmissible (MOREL), but it also frequently leads to insanity in the offspring of the drunkard (WHITEHEAD, ADAMS); and in cases where the tendency to alcoholic excesses has a hereditary origin, the cure of the dipsomaniac is generally impossible. Morel gives the following example, in which a well-marked succession of morbid phenomena became developed in different descendants of a family throughout four generations. The great-grandfather of the family was a dipsomaniac; and so complete was the transmission of the disease, that the race became totally extinct, under the well-marked phenomena of alcoholic poisoning and degeneracy.

The effects entailed were: in the first generation, alcoholic excesses, immorality, depravity, brutish disposition; in the second generation, hereditary drunkenness, attacks of mania, general paralysis; in the third generation, sobriety prevailed, but hypochondriasis, lypomania, persistent ideas of persecution, homicidal tendencies, were expressed; in the fourth generation, intelligence was but feeble, mania became developed at sixteen years of age, stupidity running on to idiocy, and to a condition involving extinction of the race. Sir James Clark also made the observation, more than twenty years ago, that the constitutions of the past three generations had deteriorated progressively from father to son (*Treatise on Pulmonary Consumption*, p. 11).

The persistent pernicious influence of the marsh poison will be fully noticed in the last part of this treatise; but there are other abundant sources of constitutionally morbid influences; and, whatever the explanation may be, it certainly is a matter of fact, as the late Dr. Alison wrote, that the *constitutional affections*, going along with the same extent of inflammation and its local effects, are extremely various in different persons previously alike in good health, or even in the same person at different times; and that we are not entitled to deny that what happens in this way in different individual cases may not happen also in nations and in seasons. As we are still very imperfectly informed as to the mode in which any local inflammation excites constitutional fever, we have no reason to doubt that the constitutional re-action consequent on the excitement of a certain degree of inflammation of the lungs may vary, equally as that which is consequent on the introduction of a certain quantity of the poison exciting *typhus fever, measles, scarlatina, or cholera*; in all of which the previous muscular strength goes for nothing in determining the degree or danger of depression or debility which may ensue.

Dr. Christison has recently communicated to the Medico-Chirurgical Society of Edinburgh his experience relative to the changes which have taken place in the constitution of fevers and inflammations in Edinburgh during the forty years previous to 1857. His experience, as well as that of many of the older physicians of Edinburgh, shows that a transition had and did every now and then take place from an *inflammatory form* of fever to one of an *asthenic type*; that it was necessary, on

the outburst of any epidemic, to watch carefully the early cases, to observe the mode in which the fatal cases terminate, and to observe generally the *constitutional tendency* or type of individual cases, in order to form an accurate judgment of the general character of the epidemic about to prevail. It will be seen also, on referring to the most approved and recent works on the diseases of India, that the descriptions of inflammations as well as fevers now seen there, when compared with the statements of Dr. Johnson, Mr. Twining, and others, twenty-five or thirty years ago, may be held to indicate that there has been a change in the usual form of re-action in inflammatory diseases in that climate as well as here. Such conclusions may be inferred from the experience of Dr. Morehead, recorded in his *Clinical Researches on the Diseases of India* (vol. ii., pp. 71, 72, and 359); and the experience of Sir Ranald Martin, expressed in his recent classic work on Climate, bears out the same observation: consult also Graves's *Clinical Medicine*, vol i., p. 303, for the doctrines of Autenrith and Graves on the change of type in disease; and for evidence regarding the deterioration of race, see p. 105 of Mr. Simons's *Third Report on the Health of Towns*.

It appears evident, therefore, that the *human body* is capable, from causes known as well as unknown to us, of undergoing various alterations as regards not only its *physical*, but also what has been termed its "*Medical Constitution*." *Fevers* are known to change their types; *epidemics* to assume new tendencies; and *inflammations* and *local lesions* to affect in no constant manner the constitution of individuals at the same period, or at different times and in different countries. This view of the subject may be summed up in the eloquent language of Dr. Watson, when he writes,—“I am firmly persuaded, by my own observation, and by the records of medicine, that there are waves of time through which the *sthenic* and the *asthenic* characters of disease prevail in succession, and that we are at present living in one of its adynamic phases” (*Edinburgh Monthly Journal*, June, 1857). It must be admitted, however, that much of what is written and here quoted regarding changes of type is based on the uncertain impressions of individual men, sometimes handed down by tradition, rather than based on the results of a deliberate deductive inquiry from statistics applied to the question by men agreed as to the meaning of terms. On the

other hand, I think Dr. Markham, in his very able Gulstonian Lectures on "The Uses of Blood-letting in Disease," puts the evidence regarding change of type in disease on much too narrow a basis—not to say an erroneous one—when he holds that it is merely an excuse put forward for a change of practice in inflammations, and that the only argument in favour of such a theory rests upon the assumed conclusion that venesection produces different effects now from what it formerly did.

A somewhat similar view of this important subject has been ably advocated by Dr. J. H. Bennett, of Edinburgh. He contends that inflammation is the same now as it has ever been—that the analogy sought to be established between it and the varying types of fevers is fallacious—that we cannot place reliance on the recorded experience of the past—and that our recent changes in therapeutics are solely due to an advanced knowledge of diagnosis and pathology (*Principles and Practice of Medicine*, p. 267).

The local phenomena of inflammation are undoubtedly constant; but the question of change of type has reference to the constitution of the individual, and to the constitutional modes by which the inflammatory state is expressed in a given number of persons. Much of the argument of Drs. Markham and Bennett appears to me to be beside the question; and there are certainly good grounds for believing that elements of constitutional degeneration exist in such abundance, especially in the communities of large towns, that Dr. Watson has, I think, happily expressed the consequence of such deterioration in the sentence quoted from his writings on the subject.

CHAPTER X.

MODES BY WHICH DISEASES TERMINATE FATALLY.

OUR knowledge on this subject is derived chiefly from three sources, namely, from the examples and illustrations afforded by the study of—(1.) Death from old age; (2.) Death from fatal injuries; (3.) The powers and actions of all our best remedies.

Such study leads to the important practical conclusion, that the same lesions of important organs may prove fatal in very different ways, and the fatal event may be averted by very different and very opposite remedies (ALISON). It is also to be observed, that in constitutions which are unimpaired, and, indeed, in every morbid process, there may be recognized a tendency to a spontaneous favourable termination.

Death happens either from the decay of life, as in old age; or it happens as an accident caused by some of those untoward lesions or derangements of the vital organs which occur in the course of the various diseases and injuries to which mankind are liable.

Death by *extreme old age* may be considered in many instances as the desirable end of a long-continued and perhaps a dreary journey. The sufferer appears to fall asleep as he might do after severe fatigue; and the long and weary journey is thus often brought to a close with little apparent derangement of the ordinary mental powers. The final scene is often brief, and the phenomena of dying are almost imperceptible. The senses fail, as if sleep were about to supervene; the perceptions become gradually more and more obtuse, and by degrees the aged man seems to pass into his final slumber. We scarce can tell the precise instant at which the solemn change from life to death has been completed. Sensation fails first, then voluntary motion; but the powers of involuntary muscular contraction, under the excitement of some external stimulus, may continue for some time longer to be feebly expressed. The blood generally ceases at first to be propelled to the extremities. The pulsations of the heart become less and less efficient. The blood fails to complete its circuit, so that the feet and hands become cold as the blood leaves them, and the decline of temperature gradually advances to the central parts. Thus far the act of dying seems to be as painless as that of falling asleep; and those who have recovered after apparent death from drowning, and after sensation has been totally lost, assert that they have experienced no pain. What is called significantly the *agony of death* may therefore be presumed to be purely automatic, and therefore unfelt. The mind, doubtless, at that solemn moment may be absorbed with that instantaneous review of impressions made upon the brain in bygone times, and which are said to present themselves with

such overwhelming power, vividness, and force, that, in the words of Montaigne, "we appear to lose, with little anxiety, the consciousness of light and of ourselves." At such a time the vivid impressions of a life well-spent must constitute that *euthanasia*—that happy death—to be desired by all.

The untoward lesions or derangements of vital organs, which occur during the progress of disease, terminate the life of man by various modes of dying. While it is ordained that eventually all must die, yet it is possible sometimes to avert, for a time, the tendency to death. To know by what agents this may be properly accomplished, it is necessary to know the modes in which death may approach in disease. Dr. Watson has happily observed that life rests upon a tripod, whose three vital supports are, the *heart*, the *brain*, and the *lungs*. Through the impaired functions of some one or more of these organs the tendency to death is expressed. The mode of dying may begin at the *head*, the *heart*, or the *lungs* (BICHAT). But inasmuch as the functions of these organs are mutually dependent upon each other, so impairment of function in any one of them may ultimately lead to death, while the mode of dying is expressed chiefly through the functions of another. The mode of dying in disease is usually a complex one, for many parts thus mutually dependent on each other are more or less immediately involved. Therefore it is of the greatest practical importance to observe how and when the different functions begin to languish, and how they may be best sustained in their exertions to maintain life.

When a person loses blood to such an extent that he faints, as from a wound, or by hæmorrhage occurring in disease, and if the flow of blood is not arrested, the state of *faint* or *syncope* continues, is not recovered from, and the heart's action ceases; not because it is unable to contract, but because its natural stimulus, the blood, is withdrawn from it, or does not arrive in sufficient quantity to be of use. This is called death by *anæmia*. In such cases, if blood can be timeously supplied to the heart (as by the operation of transfusion from a healthy person into the patient who is losing blood), the suspended function of the heart may be restored, and a supply of blood, sufficient to maintain life for a time, may be thus obtained.

The symptoms of approaching death by this mode of dying are, paleness of the countenance and lips, cold sweats, dimness of

vision, dilated pupils, vertigo, a slow, weak, irregular pulse, and speedy insensibility. If the hæmorrhage has been sudden, in large quantity, as from the uterus in "flooding," there may be nausea, or even vomiting, restlessness, tossing of the limbs, irregular sighing breathing (*anxietas*), delirium, and one or two convulsions before death ensues.

But another mode of death may be more immediately connected with the heart itself, and be independent of the supply of blood. In other words, the stimulus from blood may be sufficient, but the contractile power of the organ may fail. Such a mode of death is by *asthenia*. Many poisons act in this way, and many diseases which are due to morbid poisons in the blood tend to prove fatal by this mode of dying. Cases of extensive mortification of parts, of acute inflammation of the peritoneum, and of malignant cholera, die in this way.

The symptoms consist in the pulse becoming feeble and frequent, and ultimately failing altogether to be perceived. The muscular debility becomes extreme, but the senses remain perfect, often painfully acute, and the intellect clear to the last.

Persons whose death is by *anæmia* or by *asthenia* are often spoken of as having died in a faint, or by *syncope*; and there is still a mode of dying intermediate between the two, the type of which is seen in *death by starvation*.

Death may also be produced by the suspension of the functions of respiration, as when access of air to the lungs is prevented by a direct obstruction, either to the air-passage, in choking, or to the action of the chest, so as to prevent its expansion; or when the actions of the muscles of respiration cease, in consequence of disease or injury to the brain producing insensibility. The first of these modes by which the respiratory functions are suspended is that known as *suffocation*, technically expressed by the term *apnœa*, or privation of breath. Examples of this mode of dying may be referred to in cases of *drowning*, *smothering*, *choking*, *strangulation*, *throttling*, and closure of the *rima glottidis* by foreign bodies. In Dr. Allen Thomson's anatomical museum at Glasgow there is a larynx preserved in which a piece of coal is wedged between the *rima glottidis*. A collier thus died by *apnœa* or *suffocation* produced by the piece of coal dropping into the larynx while he lay on his back in the mine, excavating the coal from the roof of the coal pit. Forcible pressure upon the

chest, as sometimes happens in crowds during a continued crush of people, or occurs to workmen who have been buried by falls of earth and rubbish; in short, whatever causes an immovable condition of the lung-case beyond a period of three minutes and a half will thus produce a fatal result. Tetanus, and the influence of strychnine, prove fatal in this way. Morbid states produced by disease, and which terminate fatally by *apnœa*, are *œdema* of the glottis; disease of the spinal cord above the origin of the respiratory nerves (phrenic, intercostals, and spinal accessory); effusion of serum into the pleural cavities; sudden infiltration of the lungs by inflammatory exudation, or collapse of the lung in bronchitis.

The symptoms of approaching dissolution by this mode of dying are, strong but ineffectual efforts to contract the respiratory muscles, and struggling efforts to respire, amounting to agony, of short duration, followed by vertigo, loss of consciousness, and convulsions; at last all effort ceases, twitchings or tremors of the limbs alone remain, the muscles relax, and the sphincters yield. The heart and the pulse, however, still continue to act after all other signs of life are past. The recent experiments instituted by the Medico-Chirurgical Society of London show that, on an average, the heart's action continues for three minutes fifteen seconds after the animal has ceased to make respiratory efforts. On this last circumstance rests our hope of resuscitating persons so suffocated, if artificial respiration be timeously resorted to, and persevered in. This prolonged action of the heart circulates blood, which is dark, venous, and not arterialized, and accordingly the face, at first flushed, becomes turgid, and then assumes a livid and purple hue; the veins of the head and neck swell, and the eye-balls protrude from their sockets. At length the heart ceases to beat, and life is extinct (WATSON).

Death by coma occurs when there is a loss of consciousness first, with the appearance of profound sleep, from which the patient may be partially roused. The symptoms of approaching death by this mode of dying consists in a gradual blunting of sensibility to outward impressions, slowness of respiration, the inspiratory effort being often delayed, and then performed with a sudden noise and jerking inspiratory effort, technically known as *stertorous* breathing. All voluntary attention to the act of breathing is lost, but the influence of a reflex stimulus to its

performance continues. At length this function fails also. The chest ceases to expand, the blood is no longer aërated, and thenceforward precisely the same changes occur as in death by apnoea.

Such are the several modes by which death tends to approach; and “*to obviate the tendency to death*” is a doctrine which was often and strenuously inculcated by Cullen. After him, no less earnestly has it been impressed on many by my respected teacher, the late Professor Alison, whose interesting Lectures on *fevers* and *inflammation* furnished numerous illustrations. To his *Outlines of Pathology and Practice of Medicine*, and to the first volume of Dr. Watson’s *Lectures on the Principles and Practice of Physic*, the student is referred who would seek further information—sources whence the preceding observations have been mainly compiled relative to the modes by which death may approach:—

“Many are the ways that lead
To his grim cave, all dismal; yet to sense
More terrible at the entrance than within.”

“It is as natural to die as to be born;”

and thus, in

“Passing thro’ Nature to Eternity,
The sense of death is most in apprehension.”

CHAPTER XI.

PRINCIPLES WHICH DICTATE THE TREATMENT OF THE COMPLEX MORBID PROCESSES.

I. *As regards Fevers or the Febrile State.*

To avert the tendency to death in the febrile state, it is necessary to observe how fevers naturally terminate favourably. Four modes are enumerated by Dr. Parkes, namely:—

1. *By crisis*, in which the temperature falls suddenly in a few hours, and usually with some abundant excretory discharge, in which, possibly, much of the water which has been retained in the system is poured out.

2. *By lysis*, in which the fall of temperature is gradual from day to day, till the normal standard is attained. The decline

may thus occupy many days, the thermometer being known to take seven days in falling from 102° to 98° Fahr.

3. *By a combination of these two modes*, namely, by a sudden fall of temperature to a certain point, and then a gradual decrease to the normal heat.

4. Another mode has been observed by Dr. Parkes, namely, —*By a somewhat irregular alternation of febrile and non-febrile periods, as shown by the temperature and the issue.*

When *fever* terminates by any of these modes, convalescence commences, normal nutrition is renewed, and the body begins to gain in weight. The blood is poor in albumen and in red particles; and there is now a danger that the rapidity of metamorphoses of tissue will exceed the healthy standard, as shown by the great tendency which convalescents from fever have to lose heat. The temperature may fall, and the excretions may diminish below their healthy amount. Great care, constant attendance, and watchfulness, are required when the patient begins to convalesce, if the fever has been long and severe; and the treatment of the febrile state itself may be thus generally stated as consisting in a combination of measures,—(1.) To reduce excessive heat; (2.) To insure sufficient but not excessive excretion and elimination of the excretions; (3.) To act on the exhausted and semi-paralyzed nerves; (4.) To neutralize any specific poison which may have set up the fever, and so to improve the state of the blood; (5.) To relieve distressing symptoms; and lastly, To obviate and counteract local complications (PARKES, MURCHISON).

To accomplish the first indication, Dr. Robert Jackson, “the patriarch of Military Medicine,” and after him Dr. Currie, of Liverpool, practised to an extreme degree the application of cold water. In health such an application tends to increase the metamorphoses of tissue, as shown by Lehmann and Sanderson; and therefore its excessive use is contra-indicated in the febrile state. Blood-letting or hæmorrhage also tends to reduce temperature; but blood-letting can rarely be tolerated in many specific fevers, such as typhus, typhoid, scarlatina, and the like. *Infusion of digitalis* has been found by Wunderlich to have a wonderful influence in reducing and moderating the temperature in many febrile states, such as typhoid fever. Its most obvious action in small doses is to depress the force of the heart. The dose should therefore be

cautiously regulated: it must not be repeated too soon, nor be increased, if it should not operate at once.

To insure sufficient but not excessive excretion, and to promote its elimination in fever, is much more difficult than to reduce temperature; which, for obvious reasons, it is not always judicious to attempt.

The system ought to be supplied with an abundance of alkaline salts, if the urinary excretions are not eliminated. *Chloride of sodium, the alkaline salts of soda and of potash*, tend to aid the formation of urea and its elimination. Purgatives generally, and especially *salines*—*i. e.*, salts of the alkaline and earthy metals—tend to insure a proper excretion, probably by removing from the blood some of the abnormal products formed in fever, and great relief may follow their moderate use. Where urea is retained they promote its elimination, because it is known that urea sometimes passes off by the mucous membrane of the intestines. But in some fevers, as in typhus, purgatives must be very cautiously and sparingly given, and always in mild doses. So also elimination by the skin, to the extent of *diaphoresis*, is to be dreaded in typhus fever. (See “Treatment of Typhus Fever,” p. 447.)

The most important indication, however, in the management of the febrile state is to find some substance which, being “restorative” in its action (HEADLAND), will so act upon the blood, and thus restore the exhausted energies of the nervous centres. Food, mild stimulants, and quinine are all more or less employed; and quinine especially may be employed with benefit. Infusion of coffee as a medicine has been given by Dr. Parkes, with the beneficial effect of relieving headache. Böcker and Lehmann have shown that the use of coffee, in health, delays the metamorphoses of tissue, and excites the nervous system. The special treatment of the febrile state depends on the diseases of which it forms a part, and by which it is more or less modified—forming a special topic for consideration in the part which treats of special diseases. But it is above all necessary to guard against the habit of trying always to be doing *something*. As a routine system, nothing can be laid down as a rule, either in the direction of depletion, or of evacuants, or of stimulation. The febrile state is in many diseases part of the essence of the morbid state, which cannot be cut short nor materially subdued by remedies. There is no specific remedy for the cure of any fever;

and in the present state of our knowledge regarding specific febrile diseases, there can be no specific remedy for their cure.

Every disease where fever is present, and every case of specific febrile disease, must be studied so that its management or treatment may be regulated on the merits of the individual case; and must be regulated by the state of each particular function as determined by clinical investigation daily.

II. *As regards Inflammation.*

It is necessary clearly to understand and to bear in mind that, in the first instance, it is not the *lesion* which may attend the inflammatory process as a result, which is to be attended to; but it is *the diseased action tending to the lesion* which it is the object of the physician to overcome, to subdue, and turn aside; and that the occurrence of any lesion is, if possible, to be prevented. It is to the *strictly vital action—the excitement of tissue*—which tends to the organic lesion, that remedies must be applied, in order to avert the tendency to lesion, expressed by the symptoms of a constitutional kind, already referred to.

The treatment which will subdue this tendency has been technically called “*antiphlogistic treatment.*” Its mode of action depends upon the regulation and adoption of every agent, plan, or circumstance most favourable to the subsidence of the inflammation, and which will favour the influence of remedies, and oppose the advance and persistence of the inflammatory state. The treatment embraces,—(1.) Antiphlogistic regimen; (2.) Antiphlogistic remedies—means for reducing the quantity and altering the quality of the blood, and thus to counteract in a direct way the complex morbid phenomena of inflammation.

The *regimen* consists in,—(1.) A sparing allowance of non-nutritious diet; the administration of bland, simple, and cooling drinks, given often, and in small quantities; (2.) Absolute rest of body and mind; (3.) Residence in a well-ventilated apartment, maintained at an equable temperature, day and night, of about 62° Fahr.; and in cases where the lungs are inflamed, a temperature of not less than 70° Fahr. to 80° Fahr., and as dry and pure a state of the atmosphere as possible (VEALE).

The *remedies* comprehend *blood-letting, purgatives, antimonials, mercurials, opium, alkalies, and saline drugs.*

The most important and the most efficient of these remedies is undoubtedly *blood-letting*; while it must at the same time be remembered that it is not every case of inflammation that requires or warrants the abstraction of blood. It is a spoliative remedy, powerful for good and for evil. In the treatment of inflammation, it has been well observed by Dr. Watson that "each case requires its special study, speaks its proper language, furnishes its peculiar indications, and reads its own lessons." The carefully recorded facts of well-conducted though empirical observation, for hundreds of years, have attested the immediate sanative influence of blood-letting in *incipient* inflammation; and the most eminent physicians of bygone modern times have recorded, in unmistakable language, how potent is this remedy for good, and the reasons for their belief.

Our forefathers well knew when the body suffered from an inflammation in the "*inward parts*;" and in saying this we give them credit for far less scientific knowledge than they really possessed. Against such inflammations, whether in the *head*, the *chest*, or the *abdomen*, they learned, by "*watching* and not by *counting*," the sanative efficacy of early venesection; and they obtained most trustworthy evidence and experience of its power to control inflammation. Following up such doctrines will be found those veteran physicians who hold the foremost rank in the science of medicine of the present day in this country. The doctrine generally taught and universally acted upon with reference to blood-letting in inflammations is, "*so to bleed as to secure the advantages of the remedy, and to avoid its disadvantages*" (WATSON).

The standard examples of what blood-letting can do soon become apparent to every surgeon's apprentice, or hospital pupil, if he does not himself swoon the first time he sees the blood flow from the patient whom his master and teacher may wish to relieve. He may see the apoplectic sufferer roused to consciousness while the blood yet flows from the vein; and he may observe, also, that the *stounding pains* of the head in cephalic inflammations are immediately relieved, that the impatience of light and sound, the frequent, sharp, intermittent pulse, with vomiting or nausea on assuming the erect posture, the tendency to squint,—in short, all the urgent symptoms of incipient encephalitis, at once, or one by one, disappear as the blood

continues to flow. He may also notice in *thoracic* inflammation that the pain, the dyspnoea, the tightness of the chest, all disappear. Dr. Alison, as regards pleurisy, and Dr. Watson, as regards inflammation of the bowels, bear personal testimony to the good effects of blood-letting. They experienced its sanative influence in their own persons, and the practice undoubtedly saved their valuable lives from these respective diseases. Testimony from such personal experience has also been borne by the late Dr. Gregory, of Edinburgh, and before him by the celebrated Dr. Radcliffe; and so also is the testimony of many who, having experienced the benefit of the remedy once, imagine that, when again attacked with inflammation, they may be again relieved by its use. Of any one of these illustrious examples from personal experience it might be said, as Dr. Gregory said of Dr. Radcliffe, that "he was at least no fool; and we may depend upon it he would not have allowed a hundred ounces of blood to be taken from him in one day without good reason for it" (*Edin. Med. Journal*, March, 1857).

"Although much has been done," writes Dr. Alison, "particularly by the French pathologists, to enable us to judge of the texture within the chest which is the subject of inflammation, and although this is a matter of real importance, because we know that the history of the changes to be expected from inflammation in the bronchiæ, substance of the lungs and pleura, is materially different, and of course the diagnosis of these gives us a great advantage in studying the progress of any individual case,—yet as to the specific questions of blood-letting or not, the quantity, or the repetition of the blood-letting, our predecessors were very nearly as well informed as we are. It is an important practical error," he also continues, "to fix the attention, particularly of students of the profession, too much on those characters of disease which are drawn from changes of structure already effected, and to trust too exclusively to these as the diagnostics of different diseases; because, in many instances, these characters are not clearly perceptible until the latest and least remediable stage of diseases. The very object of the most important practice, moreover, in many cases, is to prevent the occurrence of the changes on which these lesions depend. After these lesions are once established, the cases are very often hopeless, or admit only of palliative treatment. In those diseases in which most can be done by art, our practice must always be guided in part by conjecture, because, if we wait for certainty, we very often wait until the time for successful practice is past; and therefore, although an accurate knowledge of the whole history

of each disease is essential to its proper treatment, yet, in a practical view, the most important part of its history is *the assemblage and succession of symptoms*, by which its nature at least, if not its precise seat, may often be known, *before any decided lesion of structure has occurred*. Accordingly, when this department of pathology is too exclusively cultivated, the attention of students is often found to be fixed on the lesions to be expected after death, more than on the power and application of remedies, either to control the diseased actions or relieve the symptoms during life."

The immediate effects of loss of blood as a remedy in inflammation are,—(1.) A sedative result on the heart's action, by diminishing the pressure on the vessels, by diminishing the quantity and altering the quality of the blood. The withdrawal of a considerable amount of stimulus from the central organ has thus a depressing effect, greater in proportion to the sudden loss of blood—the excitability of the nervous system being thus also reduced. (2.) The loss of blood generally has a derivative influence upon the blood in the part about to become the seat of effusion. This has been seen to occur in experiments upon the transparent parts of animals. (3.) Blood-letting facilitates the action of other remedies. (4.) While the effect of blood-letting is well known, by the observations of Louis and Alison, not always to check the extension of the sphere of inflammation, *yet it essentially modifies its character* (*a.*) sometimes by limitation of its sphere to a certain extent, (*b.*) by diminishing the quantity of blood from which the inflamed part is nourished, (*c.*) rendering the fibrinous exudation more liable to re-absorption.

Of late the medical profession has proclaimed with no uncertain sound, especially from the metropolis of Scotland, as to the good effects of blood-letting in the treatment of inflammation. It is well known that no remedy demands a more careful study of its application, or a more delicate adjustment of its powers; and therefore some general rules may be here stated as a guide in the use of the lancet.

1. The utility of blood-letting varies with the variations in the type of disease. A change in the type of inflammatory diseases (*i.e.*, in their usual symptoms, local and general, in their tendencies to certain local and general results rather than others, or a change in their mode of tending to a fatal termination) demands a new

study and fresh adjustment of the remedy in each particular *case, country, locality, and epidemic.*

2. There is no truth, perhaps, in medicine more conclusively determined than that we ought not to bleed, or, if we do so, we must bleed sparingly, when the inflammation depends on or is associated with the action of a morbid poison. In epidemics, therefore, of every kind, we should not hastily have recourse to the lancet, but should remember that the disease probably depends on a poison, has a course to run, and is not amenable to the mere abstraction of blood.

3. It is necessary to observe carefully, and to watch the *combination and succession* of the *constitutional and local* symptoms from the commencement of the febrile attack, and so to judge as to the propriety of blood-letting.

4. In order to obtain the good effects of the remedy by a full bleeding, it must be done *prior* to *fibrinous effusion* or *new growth*; seeing that it is *the diseased action which tends towards the lesion*—namely, effusion or growth of new material—which the physician desires to control by this remedy.

5. When the symptoms of *inflammatory fever* are little complicated and seen early, in persons previously healthy, the more violent the symptoms are, the more intense and rapid the constitutional re-action, if it does not indicate exhaustion; and the more decided the change on the function of the part affected, the more confidently we may depend on the effect of full blood-letting in relieving them.

6. When the symptoms of inflammatory fever have been uncertain and insidious in the beginning, so that the early stage has passed over unchecked, or modified by previously existing constitutional disease, or complicated with organic local disease, or when they denote debility, exhaustion, or the so-called typhoid state, they generally prove improper cases for blood-letting even when seen within the first few days.

7. Generally, it may be stated, that when the fever is high (above 104° Fahr.), when we may be sure that over a part of the inflamed organ there is congestion, stagnation of blood, distension of vessels, commencing extravasation, and change of the constitution of the blood—but these latter changes still partial and not far advanced—the power of blood-letting to control the disease has been clearly established.

8. The nature of the membrane or organ affected must always be considered in estimating the propriety of bleeding. If a serous membrane, for instance, be actually inflamed, the patient, for the most part, bears bleeding well, and is usually greatly relieved by it. With respect to organs, it is found that inflammation of the brain is less influenced by bleeding than inflammation of the liver, and inflammation of the liver than inflammation of the lungs. The gist of the evidence brought forward in Dr. Markham's very able and interesting Gulstonian Lectures establishes the special efficacy of venesection in those inflammations which are accompanied with obstructions of the cardiac and pulmonary functions, but do not prove it to be useless nor injurious in the cure of acute inflammations generally, if its remedial functions are properly used, and the advantage gained be duly followed up by appropriate remedies, such as are to be presently noticed. The symptoms which demand a full blood-letting in pneumonia are also those which indicate the greatest danger—namely, violent pyrexia, usually beginning suddenly, with full, strong, hard, and quick pulse—urgent *dyspnœa*, even *orthopnœa*—swelling and flushing of the face, frequency and violence of cough, with scanty or truly pneumonic expectoration, aggravating the pain which extends through the chest,—when such symptoms are seen within *three* days of their commencement, especially in those of robust and full habit in the prime of life, blood-letting is the remedy to be used,—everything else is *trifling*, and it is *not safe* to dispense with it. The nearer a case answers this description, the more sure we may be that the effect of blood-letting will be satisfactory, and its repetition, if the symptoms shall recur, will be well borne (ALISON). But if the inflammation be so limited that it produces little or no appreciable impediment to the respiratory or cardiac functions, when the temperature does not exceed 104° Fahr., when the pulse does not exceed 120, and when the respirations do not exceed 40 in the minute, such cases of pneumonia will get well by antiphlogistic regimen alone.

9. It is found in practice, also, that this most powerful of therapeutic agents in the cure of inflammation requires the greatest caution in its repetition, for there is a line beyond which bleeding becomes destructive instead of remedial. Two indications are of great use in determining as to the propriety of a second blood-letting, namely,—

(a.) *As to how the first bleeding is borne*—a test first suggested by the late Dr. Marshall Hall. If much blood flow from an ordinary-sized opening, before any tendency to syncope manifests itself, venesection is then considered to be well borne; if, on the contrary, the patient soon faints after a vein is opened, the judicious practitioner desists from further depletion. The urgent symptoms, being thus relieved for the moment, may again return, after a longer or shorter interval, and thus demand a repetition of the remedy, to be now judged of (b.) by the re-action of the system generally, as indicated by the state of the local symptoms, their urgency for relief, the character of the pulse, and the appearance of the blood first drawn. The re-action may be of such a kind that a *sthenic* state of *inflammatory fever* still continues, or returns after temporary subsidence. The inflammatory process having been interrupted, so far modified, but not arrested, the remission proves transient, and the re-accession may be more fierce than the onset. A repetition of blood-letting is demanded so soon as such re-action has declared itself. On the contrary, the re-action may be *asthenic*, or of nervous character, the pulse being rapid, soft, and jerking, the breathing oppressed, headache and *tinnitus aurium* present, with general nervous excitement; bleeding, under such circumstances, *is not to be repeated*. A full opiate will allay the nervous excitement.

10. The next consideration is, "What indications for bleeding are to be drawn from the state of the blood?" The blood offers certain indications, either for bleeding or refraining from it, when the symptoms would otherwise demand or forbid this operation. The firmness of the coagulum, for example, has been considered at all times as a mark of the tonic state of the system, and as a warranty for repeating the bleeding when the part is as yet unrelieved, and the re-action continues of the *sthenic* type.

The thickness, and especially the firmness, of the *buffy* coat, if lifted on a pin, was one of the leading characteristics of the existence of acute inflammation, amongst others already noticed, and was much founded upon by Dr. Gregory, as guiding his practice in the treatment of inflammation. On the contrary, a looseness of texture of the clot is a sure sign of great debility, so that, unless other circumstances strongly indicate the necessity of bleeding, it ought not to be repeated when the yellow or buffy substance on the surface is loose and not thick.

11. The proportion of the serum to the clot, and also its occasionally altered characters, are arguments for or against bleeding. When the quantity of serum is unusually large, unless the clot be very firm, bleeding ought not to be repeated. Also, when the properties of the serum are so altered that it coagulates and forms one mass with the clot, bleeding is constantly prejudicial; and, lastly, it has been observed that when the serum, which has little or no affinity for the red globules in health, readily dissolves them, it is an unerring sign that further bleeding should be avoided. In some of the febrile diseases the fibrin never augments, remains often in normal quantity, and is also often diminished. In the acute inflammatory fever, on the contrary, there is a constant augmentation of this principle, compared with the red globules, as observed by Andral. It is this excess of fibrin which gives firmness to the clot, and is the cause of its being "buffed" and "cupped." The immediate effect of bleeding, according to the same high authority, is to reduce the red globules, but not so with the fibrin; for a reduction of the fibrin does not take place till after a certain time. Such is the state of the blood in the sthenic inflammatory states. There are many reasons, however, for not esteeming the buffed and cupped state of the blood, denoting an excess of fibrin, as a sufficient warranty for bleeding; for these conditions are often present in erysipelas, phthisis, or the early stages of typhus fever; and in either of these cases the loss of a moderate quantity of blood might hurry the patient to his tomb. Again, in acute rheumatism the blood is not only buffed and cupped, but contains a maximum quantity of fibrin; yet the best practitioners seldom think it necessary to take blood, considering that mode of treatment as neither affording present relief nor shortening the course of the disease. The fact, then, of the blood being buffed and cupped does not in all cases warrant venesection. It is also well known that the sthenic or buffed characters of the blood are often greatly modified by the manner in which the blood is drawn; thus, if an individual be bled in both arms, and the blood allowed to flow with different velocities—that is, in a full stream from one and slowly from the other—the blood drawn is identically the same; yet a thick buff will be wanting in the latter and be present in the former. And if the apertures be of different sizes, the same differences will result; the blood

from the larger orifice will be buffed, while no such effect is seen in the blood drawn from the smaller one. Again, the form of the vessel which receives the blood, as to whether it be flat or conical, and also its temperature, or whether the blood be received into one that is cold or warm, will also affect the phenomena of its coagulation.

There are many circumstances, therefore, which prevent the blood from being an unerring guide for bleeding in cases of inflammation; but *the assemblage and succession of symptoms* must decide as to the propriety of blood-letting in doubtful cases.

12. An improvement in the character of the secretion or excretion from the inflamed part contra-indicates the repetition of blood-letting; for instance, in pneumonia, if the *character* of the expectoration, from being scanty, tenacious, and tinged with blood, becomes copious and free, much may be expected from this natural tendency to cure.

13. It is an object to effect the sanative result with as little expenditure of blood as possible; but the amount to be taken can only be judged of by the effects produced. The patient should be bled, if possible, in the upright position, and a full stream of blood allowed to flow from a sufficiently large orifice in a vein. To accomplish this fully, it may sometimes be necessary to open a vein in each arm, so that the flow may be from both at the same time.

Blood-letting may be employed either generally or locally. General bleeding is best adapted to subdue acute inflammation of visceral organs, because it makes a more decided and rapid impression upon the system. Local blood-letting, by leeching, scarification, or cupping, is more useful in chronic inflammation, and in inflammations affecting membranes, such as the pleura, peritoneum, meninges, and articular membranes; but it is often advantageous to combine the two methods of taking blood. In the case of external visible inflammation, the direct abstraction of blood from the inflamed part during the congestive period of the inflammatory process, is seen to exercise a benign influence over the progress of the inflammation.

The next most important class of antiphlogistic agents in the treatment of inflammation consists of *purgatives*, especially the *resinous cathartics*, like *scammony* and *gamboge*. (1.) They free the stomach and intestines from accumulated food and fæces, or

other irritating and acrid matters. (2.) They subdue the inflammatory tendency by the discharge of a large quantity of serous fluid charged with albumen, from a large extent of mucous membrane. Thus they tend to check effusions, and diminish the force of the heart's action. Their use is especially indicated in *encephalic* inflammations and *hepatic* congestions; but they are less efficient in subduing *thoracic* inflammation; while in the *enteric* inflammations they ought not to be pushed beyond merely unloading the alimentary canal. Combined with diaphoretics, they promote the elimination of morbid material through the glands.

The influence of mercury varies with its mode of administration and the constitution of the patient. It is followed, in large doses, by an increased flow of watery evacuations from the bowels, and an increased flow of saliva.* If the use of the remedy is continued, especially in small and repeated doses, combined with opium, so that it is not passed off by the bowels, this mineral induces *salivation*—that is, saliva flows profusely, the gums become tender, red, swollen, and ulcerated on the margins in contact with the teeth. The patient gets rapidly thin during its use. The blood is decomposed, the red corpuscles are rapidly destroyed to the extent of *one-sixth* or more; the fibrin is diminished by *one-third* of its amount; the albumen by *one-seventh*; and, at the same time, it may become loaded with a fetid matter, the product of decomposition (WRIGHT). “Thus it is an agent of terrible activity, and we may well be cautious how we handle it” (HEADLAND). Its sanative power is believed to consist in controlling or preventing the coagulation of lymph; and for this purpose it is used as an auxiliary to blood-letting. It is only useful in the *sthenic* forms of inflammatory action. It is decidedly hurtful in cases of erysipelas disposed to gangrene, in scrofulous states of the system, in debility, and in cases where the nervous system is in an irritable condition, and the condition of the patient tending to the so-called typhoid state. The specific

* The experiments of Dr. George Scott, of Southampton, throw considerable doubts on the hitherto generally received opinion, that calomel in large and purgative doses increases the flow of bile; on the contrary, such doses seem, in the first instance, to diminish the flow of bile; and it is a matter for further experiment to determine whether small and frequent doses of calomel, continued for a length of time, so as to produce the specific action of mercury upon the system, will really ultimately augment the biliary secretion (Beale's *Archives*, vol. i., p. 209).

influence of mercury is recognized by the tenderness of the gums which it induces, by the increase in the quantity of saliva, and by the peculiar mercurial fetor of the breath. This is the utmost action which should ever be induced. Calomel alone, or calomel combined with opium (a quarter of a grain of the latter with two grains of the former; or a third of a grain of opium with three or four grains of calomel), every three, four, or six hours, is the best form of administration where its influence is rapidly required. But this form of combining mercury with opium should not be persevered in too long, otherwise copious salivation may be induced, which, of all things, ought to be avoided.

Mercury thus employed tends to prevent effusion and favour absorption of effused products. It is advantageously employed in membranous inflammations, and such as go on slowly.

Antimony is antiphlogistic, by tending to increase all the secretions; but particularly those from the skin and lungs. It is especially useful in those sthenic inflammations which are rapid, and in which a sudden and powerful action is desired; and also where the direct sanative influence of blood-letting is to be maintained. It is thus indispensable in *croup*, extremely efficacious in *sthenic pneumonia*, and highly useful in *bronchitis*. As an agent to keep up the sanative influence obtained by blood-letting, the action of antimony is invaluable; for when blood-letting has weakened the force of the heart, by diminishing the pressure on the vessels, then antimony maintains this diminished pressure in proportion as it weakens the force of the heart. A perseverance in its use produces a watery condition of the blood, diminishing especially the amount of fibrin. The production of nausea is an indication that it has taken sufficient effect.

Alkalies dissolve the fibrin of the blood and retard its formation; and the ingredients of the urine produced out of the destruction of the albuminous compounds of the body are increased largely in amount by the administration of alkalies. They generally pass out of the body as salts, having combined with acids in the system, and tending to leave behind them an excess of alkali in the blood.

All treatment ought to be judiciously regulated by the knowledge of the tendency of the disease to a spontaneous favourable termination—the accidental symptoms of urgency requiring treatment and control in many cases, rather than the disease itself.

THE

SCIENCE AND PRACTICE OF MEDICINE.

PART II.

METHODICAL NOSOLOGY—SYSTEMATIC MEDICINE, OR THE DISTINCTIONS AND DEFINITIONS, THE NOMEN- CLATURE AND CLASSIFICATION OF DISEASES.

CHAPTER I.

THE AIM AND OBJECTS OF NOSOLOGY.

NOSOLOGY, regarded as a distinct department of the Science of Medicine, embraces three separate objects of consideration—namely, *First*, the DISTINCTION and DEFINITION of particular diseases, or of the genera and species of diseases; *Secondly*, The NOMENCLATURE of diseases, or the assignment of the names by which they are to be designated, so that each disease may be distinguished and known by an appropriate name; and, *Thirdly*, The ARRANGEMENT or CLASSIFICATION of diseases in some methodic and convenient order, by which they may be distributed into classes, orders, genera, or species. These three divisions of Nosology are respectively known as the DEFINITION, the NOMENCLATURE, and the CLASSIFICATION of diseases. Of these in their order.

I. *The Definitions of Diseases.*—The first object of Nosology is to obtain such brief enumerations of the peculiar characters of diseases as are sufficient, provisionally, to define them; and the practice of attempting to define diseases so as to lead to their

being easily recognized was begun before the time of Galen. In modern times the great advantages that have arisen from establishing definitions in natural history upon fixed and determinate principles, not only of its various objects individually, but also of the groups under which it was found possible to arrange them, suggested to medical men the idea that much advantage might also result to the Science of Medicine from defining diseases, and such groups of diseases as might be found expedient to recognize, under general terms or common names, based upon some fixed and determinate principles. Sydenham recommended that definitions or brief descriptions of diseases should be framed after the model of those that are given of plants; and he lays down various judicious rules for the proper execution of this object in the preface to his work *On Acute Diseases*, first published in 1675. The precepts of Sydenham were never reduced to practice in his day; but about fifty-seven years after his work *On Acute Diseases* was published, the idea was taken up and acted upon by Franciscus Boissier de Sauvages, a distinguished physician and eminent professor of medicine at Montpellier. He attempted to arrange diseases, as botanists have done plants, into classes, orders, and genera. He endeavoured to lay down the characteristic phenomena of each, and to enumerate their principal varieties. The outlines of his nosological system were first published in 1732, and followed thirty years afterwards by his *Nosologia Methodica*—a work which marks an important era in the history of Medicine, as having led to much greater accuracy in the distinction of diseases than was previously observed.

At present the only useful method of defining diseases seems to be an artificial one. It is assumed by nosologists that the proper foundation for the distinction of particular diseases is the occurrence of constant and uniform combinations of morbid phenomena or symptoms, presenting themselves in concurrence or in succession. Thus some of the essentials of a definition are obtained, so that each disease may be marked out by such a brief enumeration of its leading characters as might serve to distinguish it from every other. A series of nosological definitions, more or less correct, may be thus provisionally established, so that the same things are designated by the same terms. Objections have been urged to methods of this kind,

on the ground that diseases are unsteady and variable in their character; but the aids to science are now so numerous that physicians are becoming more and more able to distinguish diseases from one another, and to tell by what marks, or upon what grounds, they do so distinguish them. Such are the marks or grounds of distinction by which each disease ought to be defined; and as often as we attempt to establish a distinction among diseases, either the deficiencies or the errors of our definitions will be the more easily perceived; and the attempt will lead to a more accurate consideration of observations previously made, as well as to a greater degree of accuracy in subsequent observations. Definitions of diseases are therefore not only of much service to methodical nosology, but they help to render the diagnosis of diseases more perfect. Pathologists, however, are not agreed as to whether the definitions of diseases should be derived from the external phenomena that present themselves in their course, or from the internal pathological conditions on which these phenomena are supposed to depend; and particularly such of these conditions as consist in lesions or structural alterations discoverable after death.

Cullen was in favour of definitions derived from the symptoms; but he believed that the information derived from pathological anatomy might guide to correct distinctions among diseases. Defining diseases by their supposed proximate causes may lead to error, inasmuch as in many cases these causes are disputable, and may long continue to be so. Whatever principle of defining diseases be adopted, it is absolutely necessary that it should be independent of every theoretical view; for any theory employed, however specious, however much we may be persuaded of its truth, may not appear in the same point of view to others, and may therefore occasion endless confusion (CULLEN). If no uniform principle can be laid down for arriving at precise definitions of disease, we must be content with such methods of definition as will serve the main purpose of coupling intelligible general notions regarding the disease with given modes of expression. For example, although we cannot give such a definition of many a disease as will embrace even all the leading phenomena of every case, we may assuredly give such a definition as shall apply with reasonable accuracy to the disease we intend to designate, so that no one may suppose we mean thereby either

small-pox or the *gout*, when we mean *typhus fever* or *dysentery*; and, in a progressive science like medicine, definitions must always be provisional.

II. **The Nomenclature of Diseases.**—This, the second object of Nosology, has given rise to many disputes, and has furnished much scope for the display of classical erudition. From the earliest periods of medicine the names imposed upon diseases have been derived from several different sources; but the following considerations have generally regulated the naming of a disease:—*First*: Some names have been taken from the part affected—*e. g.*, *peripneumonia*, *podagra*, *ophthalmia*, *dysentery*. *Secondly*: The most characteristic symptoms have furnished the name—*e. g.*, *ileus*, *tenesmus*, *paralysis*, *diarrhœa*, *dyspnœa*, *coma*. *Thirdly*: Some names have been taken from these two circumstances combined—*e. g.*, *cephalalgia*, *otalgia*, *cardialgia*, *odontalgia*, *hysteralgia*. *Fourthly*: An alteration of tissue upon which subsequent changes depend being recognized as the essential element of the disease, it is named accordingly—*e. g.*, *pleuritis*, *peritonitis*. *Fifthly*: Such alteration not being discovered, the first tangible link in the chain of causation has been used instead—*e. g.*, *melancholia*, *cholera*, *typhus*. *Sixthly*: When a lesion tending to sudden death at once follows the application of a cause, that cause may name the disease—*e. g.*, *lightning*, *prussic acid*, *arsenic*, *burn*, *scald*, *sun-stroke*, *cut*, *stab*, *frost-bite*, &c. *Seventhly*: A considerable number of names of diseases have been derived from some imaginary resemblance to external objects—*e. g.*, *elephantiasis*, *cancer*, *polypus*, *anthrax*, &c. *Lastly*: There are still many names the origin of which it is not now easy to trace.

It is obvious, from these statements, that the names of diseases must change as our knowledge changes and becomes more precise; and many diseases which were once named after their symptoms are now called according to the lesion from which most of those symptoms proceed. An apt illustration of this is to be found in *paralysis*, which is no longer regarded as a disease *per se*, but is merely a symptom of several structural alterations of the brain and spinal marrow; and so also *diarrhœa*, which now ought to be almost excluded as a disease from tables of the causes of death.

The progress made in our knowledge of disease from time to time rendered it obvious that some diseases, now only sufficiently recognizable, are different from any other diseases hitherto known.

In separating them it became necessary to invent new names for the distinct diseases, or a choice had to be made from amongst those names previously in use. Hence the jumble of Greek, Latin, and mongrel names which pervades medical nomenclature. The idea also of rendering medical nomenclature *uniform*, by deriving the names of diseases from one source only, or from a certain or mixed combination of sources, has caused many to attempt the reform of medical nomenclature, and especially since morbid anatomy has been so much prosecuted that it might serve as a useful guide in distinguishing the disease or dictating its name.

By some it is maintained that "the name of each disease or species should be so characteristic and significant that a person slightly acquainted with the language and the subject should, on hearing it, immediately understand what is the nature of the disease it designates" (PLOUCQUET). In this respect the name ought to be composed out of the same elements as the definition of the disease—in fact, it ought to be the definition converted into a name, and derived either from the symptoms of the disease or from the supposed proximate cause. But a name which is expressive only of the nature, seat, or proximate cause of a disease may be erroneous in respect of each of these facts singly, or of all of them together. The history of the nomenclature of fever, especially typhoid, would amply illustrate these statements—*e. g.*, putrid fever, adynamic fever, bilious fever, pythogenic fever, enteric fever, meningo-gastric fever, nervous fever, gastric fever, are mild examples of nomenclature and of confusion which ought to make a man pause before he attempts to construct a new name. It is inexpedient, also, to abandon (except when unavoidable) the names of distinct diseases received and recognized by our forefathers in the science; or of substituting new ones in their place, without an extreme necessity. Sauvages insists much on this point, and Cullen was of the same opinion. "Words," says the former, "are good only in respect of their signification." In dealing, therefore, with ancient nomenclature, which, for the time being, may appear objectionable, it is surely better to extend, if possible, the signification of the word, name, or term, than to alter it. At the same time it must always be permitted to give new names to new diseases, and to select the best out of those which are in use, when a great number have been used to designate one and the same thing. There are some

principles, therefore, which it is well to recognize as influencing the judicious choice of a name. Such names, for example, as involve or attempt to indicate a proximate cause are more liable to lead to error than those which are derived from leading symptoms. If names were to be based on supposed causes, new names of diseases would be required whenever a new hypothesis is started. Look, for example, at the names of typhoid fever, already mentioned, and the systems of Linnæus, Vogel, Pinel, and even Mason Good, will show that medical nomenclature has been repeatedly changed without any urgent necessity; and great inconvenience has especially resulted from incorporating particular and often peculiar pathological doctrines with the language and nomenclature of diseases. So much has this been the case that the language of medical science has been in danger of becoming "a curious mosaic of the chief speculations of ancient and modern times." The passion for inventing new terms retards also, in a wonderful degree, the progress of the student of medicine, and tends to involve him in difficulty and doubt.

III. The Classification of Diseases.—From time to time physicians have considered it advisable or advantageous to arrange the whole of the diseases they are able to define, and to name, under more or less comprehensive groups. A consideration of the different plans which may be pursued in such arrangements, and of the advantages to be derived from them, forms the third object of Nosology.

It is obvious that any single character, or combination of characters, in respect of which diseases agree with or differ from each other, may be made the basis of methodical arrangement, under a larger or smaller number of divisions, or of higher or lower genera (language of logicians), or of classes, orders, and genera (language of naturalists). By ingenious devices of the mind the physician or the statist may classify and arrange his knowledge so as to bring it all more readily within his reach for any special purpose,—so as to make it, in fact, more at his disposal—to facilitate and pave the way for further investigation. Such are the legitimate objects and the results of all methodical arrangements. Classification, therefore, being only a method of generalization, there are, of course, several classifications of disease which may be used with advantage for special purposes. The

physician, the pathologist, the jurist, the hospital statist, the army medical officer, may each legitimately classify diseases from his own point of view, and for his own purposes, in the way that he thinks the best adapted to facilitate his inquiries, and to yield him general results. The medical practitioner may found his main divisions of diseases on their treatment, as medical or surgical; the pathologist, on the nature of the morbid action or product; the anatomist or the physiologist, on the tissues and organs involved; the medical jurist, on the suddenness, slowness, violent, or unnatural mode of the death; the hospital statist, on the kind of diseases which are treated in its wards; and all of these points of view may give useful and interesting results (FARR).

There is thus no question on which more diversified opinions are legitimately entertained than on that of classification. Although it is the aim of all systematic writers and observers to arrange the objects of study in the most natural order possible, and although diseases are named as if they were individual entities, yet they present so great varieties that they will not admit of that definite and, in many respects, natural species of classification which can be made with objects of natural history. Manifest reasons of convenience and facility for work can therefore be assigned as the great incentive to classification; and numerous reasons exist for classifying diseases in various ways:—(1.) Men differ in their estimation of the characters on which different arrangements may be founded. (2.) The facts and phenomena of diseases on which classifications may be made are not all regarded from the same point of view. Most systems are avowedly *artificial*, being arranged with the view to elucidate or support a theory, or otherwise to effect a definite end. For example, by classifying diseases and recording the causes of death, the most valuable information is obtained relative to the health of the people, or of the unwholesomeness and pestilential agencies which surround them. “We can take this or that disease, and measure not only its destructiveness, but its favourite times of visitation; we can identify its haunts and classify its victims.” We are able to trace diseases also as they perceptibly get weaker and weaker, or otherwise change their type, as some have done from time to time. We know from the valuable returns of the registrar-general, prepared periodically by Dr. Farr, that certain diseases are decreasing, or growing less and less destructive; that certain

other diseases have ceased in some measure; while other severe diseases have exhibited a tendency to increase. The advantages, therefore, of adopting some system of classifying diseases, which can be put to such useful practical purposes, must be obvious to every one.

To some extent other systems are *natural* in their arrangement, in so far as they attempt to express or exhibit some of the natural relations which subsist among diseases; but the mere expression of one man's interpretation of peculiarities of disease of the same species, and the elevation of such diseases in a classification as specifically distinct, are apt to be based on insufficient evidence as regards natural relations.

Principles of Classification.—Many systems of Nosology have been adopted from time to time; and as valuable general principles have been adduced from some, the grounds on which diseases have been classified may be briefly described under the following nine divisions, namely:—

I. *The nature of the ascertained causes of disease.* On this principle two classes of diseases are recognized, namely,—(1.) Diseases arising from general causes; (2.) Diseases arising from specific causes.

II. *The pathological states or conditions which attend diseases.* The principle of this classification consists in determining alterations of the structure or the chemical composition of parts, from which names are given to the disease—*e. g.*, pleuritis, pneumonia, &c. The distinctions of Sauvages were generally derived from symptomatic and pathological characters, or external symptoms alone; Cullen, following (1792), adopted similar grounds of classification; but with much more comprehensive views than Sauvages, a more lucid order, and a happier simplicity, he excelled in accuracy of definitions all who had gone before him. His descriptions of disease received no colouring from his theories. They are faithful to nature, consistent with the knowledge of his day; and, greatly in advance of his time, his original and inventive mind dwelt much on the causes of disease in all his reasonings and explanations on medical subjects. Aware, however, of the imperfections of the Art of Medicine, he did not attempt to arrange diseases according to their proximate causes, but according to a method founded partly on their symptoms, partly on their causes, and partly on their seats (CURRIE). A methodical arrangement

of this kind has generally been considered the most desirable, as being likely to bring together diseases corresponding not only in some very important relations as regards their symptoms, but also in the indications and means of treatment which they suggest and require. But it is obvious that such an arrangement must vary according to the progress of knowledge and of opinion; for a disease which may at present be supposed to depend upon one pathological condition may be found at a future time to proceed from another. Besides, the arrangement involves a principle which tends to separate diseases bearing a striking resemblance to one another in their external phenomena, though depending on different pathological conditions; for example, different species of apoplexy and epilepsy. It is an arrangement, also, which brings together diseases which, though belonging to the same natural family, may be respectively characterized by groups of symptoms that do not bear any very obvious resemblance. Thus the hæmorrhages at once bring together apoplexy and hæmoptysis in this classification.

III. *The properties, powers, or functions of an organ or system of organs being deranged*, dictates a classification in which the most prominent effects or phenomena of morbid states are considered as the disease—*e. g.*, palpitation, diarrhœa. It is an arrangement which brings diseases into approximation with one another according to the part of the body principally affected and the function principally disturbed.

When disease consists in perverted powers or functions, it is then denominated a *dynamic* affection or disorder. When it depends on change of structure, it is termed an *organic* lesion or disease.

This third basis of classification is Physiological, and was adopted by Drs. Young and Mason Good in imitation of Ploucquet, of Tübingen. It has been the most popular arrangement of diseases, and perhaps the best adapted for lectures, or for treatises on the practice of physic, because it brings together the different diseases of the same organ, and of those organs most intimately related to one another; but, to profit by the arrangement, the student must be previously instructed in the general doctrines of disease.

IV. The diseases comprehended under the two latter principles of classification are sometimes inaccurately and loosely brought

together under the heads of *Structural* and *Functional* diseases. The diseases of function, for instance, being made to embrace the *neuroses*, *hæmorrhages*, and *dropsies*; while *inflammation*, *tubercle*, *cancer*, *melanosis*, *hypertrophy*, and *atrophy* are the subordinate classes of the diseases of structure. The diseases of function embrace all those diseases in which the action, the secretion, or the sensation of a part is impaired, without any primary alteration of structure of the organ or tissue affected, so far as our imperfect means of research can ascertain. Thus, *mania*, *catalepsy*, *neuralgia*, are *neuroses* of the brain or other portions of the nervous system. *Colic*, *vomiting*, *diarrhœa*, and *constipation* are *neuroses* of the alimentary canal; and so on of other parts. *Hæmorrhage*, or the effusion of blood, and *dropsies*, or an effusion of water into the shut cavities of the body, as that of the head, chest, or abdomen, are also instances of functional disease. Such are the grounds of classification adopted by the late Dr. Williams, of St. Thomas's Hospital, London.

V. A basis of classification has been adopted, founded on *the pathological nature of the different morbid processes*, but the arrangement of the orders and subdivisions are determined by the anatomical arrangement of the textures and organs of the animal body, as originally developed by Bichat.

Such is the principle and mode of classification adopted by Dr. Craigie (1836).

VI. A ground of classification exists, having reference to *the general nature and localization of the morbid states*. It comprehends three classes,—(1.) Diseases which occupy the whole system at the same time, and in which all the functions are simultaneously deranged. These have been named general diseases, such as *fevers*. (2.) Constitutional affections, meaning thereby diseases which display themselves in local lesions in any part, or in several parts of the system, but not in all parts at the same time—*e.g.*, *rheumatism*, *gout*. (3.) Local morbid processes.

Such is the classification adopted by Dr. Wood, of Pennsylvania (1847).

VII. Applying the principles of a *purely humoral pathology*, we have a classification consisting of,—

a. Fevers. b. Dyscrasiæ—*e.g.*, *tabes*, *chlorosis*, *scorbutus*, *dropsy*,

diabetes, pyæmia, tuberculosis, carcinoma. c. Constitutional diseases, induced by,—(1.) Specific agents; (2.) Vegetable substances. Such is Wunderlich's arrangement of diseases (1852).

VIII. M. de Savignac, Professor of Clinical Medicine at the Naval School of Toulon, propounded (1861) a Nosological arrangement founded on what he believes to be the "elements" of disease. To each of the classes he so defines, the question would at once suggest itself, and require solution, as to what the "element" may be on which the particular class is made to stand alone. He merely subjoins the word "element" to an adjective formed from the name of each class of diseases. Thus the class Neuroses is distinguished by the *neurosic element*; the class Rheumatalgiæ, by the *rheumatic element*; and so on to the number of fourteen classes. In the formation of orders, genera, or groups of diseases under this classification, no fixed principle can be recognized.

IX. Dr. Stark, of Edinburgh, proposed (1864) an arrangement embracing sixteen classes, namely:—(1.) Fevers; (2.) Diseases of the brain, &c.; (3.) Diseases of the heart and organs of circulation; (4.) Diseases of organs of respiration; (5.) Diseases of organs of digestion; (6.) Diseases of urinary organs; (7.) Diseases of organs of generation; (8.) Diseases of organs of locomotion; (9.) Diseases of skin and cellular tissue; (10.) Diseases of uncertain seat; (11.) Malformation; (12.) Debility at birth, and premature birth; (13.) Old age; (14.) Sudden deaths; (15.) Violent or unnatural deaths; (16.) Causes not specified.

None of these nine methods lead to a perfectly philosophical or purely natural classification, because diseases are not yet sufficiently understood to permit us to see clearly their mutual relations; and the best recommendation of any one of them would be a negative one—namely, that of doing the least possible violence to our very imperfect knowledge regarding the natural affinities or alliances of diseases, of which we have at present only a sort of instinctive recognition. But the tendency of modern investigations by the varied instruments and methods of research tends to prove that many diseases hitherto supposed to be altogether functional are really accompanied with changes of structure, of an anatomical, physical, or chemical kind. It is therefore not unreasonable to anticipate that all the so-called functional maladies will be found to depend upon some concomi-

tant alteration of structure; and when we are unable to detect an alteration either of the solid or fluid parts of the body, in cases where the existence of disease cannot be doubted, we may attribute our failure to the imperfection of our means and instruments of observation, or our modes of using them.

In the present imperfect state of our knowledge, therefore, diseases cannot be philosophically classified, nor arranged according to natural or true pathological relations, dependencies, or alliances. Nevertheless, a great advantage inevitably results from the institution of nosological classification, on account of the necessity which every such attempt imposes on those who engage in it, of marking very accurately the characteristic phenomena of particular diseases; and every one acquainted with the progress of natural history must know that the study of details, and the repeated attempts to systematize them, have mutually promoted and supported each other. It is the same with regard to diseases; and if a Methodical Nosology cannot be rendered perfect, it is a certain proof that, for the time being, the details of which it must be composed are neither accurate nor complete, and are not likely to be so till attempts to observe, investigate, and systematize have made some further progress. Every attempt to reduce to system tends to enlarge our stock of facts; and though we may fail to obtain a perfectly philosophical arrangement, yet the very attempt to attain it must be of advantage, by leading to useful discussions regarding the Pathology and History of diseases (CULLEN). No one could be more convinced than Cullen was, that "perfect division and definition is the summit of human knowledge in every department of science, and requires not only the clearest, but the most comprehensive views, such as (with respect to diseases) we can arrive at only by often repeated attempts and much study." A no less distinguished pathologist—M. Bayle—in discussing the difficulties connected with classification, recommends us "to follow the plan which presents fewest imperfections, remembering that the determination of specific characters is what is most essential in Nosology, arrangement being the least important; for each arrangement will have its defects, will present its deficiencies, and exhibit some forced approximations." Every plan of arrangement ought, therefore, to be accepted for what it is worth, and appreciated at its true

value; namely, as to how far it fulfils the object for which it was mainly devised. Cullen, also, in his lectures and in his writings on this subject, everywhere speaks with the utmost modesty and diffidence, and endeavours at all times to impress upon the mind the fact that Nosology, like other branches of medical science, must necessarily be progressive in its advancement; and that it is only by frequent and multiplied trials that it can be brought to any degree of perfection. A perfectly philosophical or natural system of classification aims at having the details of its plan to agree in every respect with all the facts as they exist in nature. To effect this end, arrangements, as they *naturally* exist, require to be traced out, not devised. The tracts in which such a pursuit must be followed up, and in which our knowledge is as yet deficient, may be shortly indicated under the following heads, namely:—(1.) The affinities or alliances of diseases with each other. (2.) The morbid anatomy of diseased parts. (3.) The communication, propagation, inoculation, generation, development, course, and spontaneous natural termination of diseases. (4.) The connection of the phenomena recognized during life with the facts of morbid anatomy. (5.) The geographical distribution of diseases. (6.) The succession of diseases, so far as they can be traced through past ages; the peculiarities they have exhibited at different periods in the world's history, or within comparatively recent cycles of years. But the time has not yet come for a classification on a basis so comprehensive—simply because the material does not yet exist; and attempts to make so-called *natural* systems of arrangement must end in disappointment, on account of the uncertain and fluctuating data on which they must be based. Such attempts are apt to suggest the serious question, “Whether such Nosology promotes or retards the progress of Medicine?”

Present State and Aim of Nosology.—The most distinguished Physicians and Statists are lending their joint aid to obtain a nomenclature and classification of diseases which can be applied to the wants of the civil and military population in every country. Dr. William Farr, devised a system of Nosology which was discussed at several meetings of the Statistical Congress of the Great Powers of Europe, convened for the purpose, amongst other business, of devising and adopting an uniform system of nomenclature for recording diseases and the causes of death from them. The Congress met in Paris on

the 10th of September, 1855, when a nomenclature of the causes of death was agreed upon, essentially the same as that used in England and Geneva. At a third Conference, held at Vienna in 1857, a *nomenclature* substantially uniform was agreed upon for adoption in all the States of Europe; and fatal cases were to be registered on an uniform plan. Dr. Farr's system of nomenclature has been in use now for many years by the Registrar-General of this country, and more recently by the Army Medical Department. It was also adopted in previous editions of this text-book; and although it had many imperfections and defects, it was still practically the most useful and authoritative Nosology. "In the English list of names," proposed and adopted by the College of Physicians, "it seemed desirable that as little deviation as possible should be made from those employed by the Registrar-General of England; otherwise his settled plans, and his forms of returns, which have been followed for years, would require to be re-modelled; the comparison of future years with past returns would be made difficult and perplexing, if not impossible; and a damaging break would be caused in evidence which becomes more and more trustworthy and valuable in proportion as it is prolonged and continuous."

The important task of devising a "Provisional Nomenclature and Definition of Diseases," consistent with the progress of medical science, has engaged the attention of a Committee of the Royal College of Physicians of London during the past ten years (1857-1867). After many interruptions and much consideration, the Committee at last completed their work, and submitted a Provisional Nomenclature to the College on the 3d of August, and again on the 28th of November, 1867, when it was unanimously adopted.*

* The circumstances of the origin and progress of this great work are of historical importance, and are thus recorded by the College:—"The idea which led to the formation of a general Nomenclature of Diseases originated in a correspondence between Dr. Dumbreck, of the Medical Department of the Army, and Dr. Sibson, respecting the need of such a nomenclature for use in the Army Medical Service. But at the *Comitia majora* of the Royal College of Physicians, held on the 9th July, 1857, it was resolved, on the motion of Dr. Nairne, in consequence of a letter addressed to the College by the Hospitals' Committee of the Epidemiological Society, 'That a Committee be appointed to prepare a Nomenclature of Diseases, and that such Committee have full power to co-operate with other bodies.'

"The following Fellows of the College were appointed members of the Committee by the President of the College:—Drs. Mayo, Alderson, Hawkins, Jeaffreson, Pit-

This is a great achievement. The Registrars-General of England, Scotland, and Ireland, the chiefs of the Medical Department of the Army and Navy, and of the British troops in India, have all concurred with others in framing the Nomenclature;

man, Bence Jones, Risdon Bennet, Munk, Babington, Addison, Nairne, Barker, Budd, Gull, Baly, Barclay, Sibson, Parkes. At the first meeting of the Committee Dr. Sibson was appointed Secretary.

“The following representative members afterwards consented to co-operate in carrying into effect the objects of the Committee:—Mr. Stanley, President of the Royal College of Surgeons; Dr. Druitt, Representative of the Master of the Worshipful Society of Apothecaries; Sir John Liddell, Director-General of the Medical Department of the Navy; Dr. Logan, Director-General of the Medical Department of the Army; Sir Ranald Martin, Representative of the East India Company; Dr. Farr, Representative of the Registrar-General; Mr. Simon, Medical Officer of the Privy Council; Mr. Holmes, Secretary of the Hospitals' Committee of the Epidemiological Society.

“The meetings of the Committee were suspended in 1858, in consequence of the passing of the Medical Act of that year, and of the alterations thereby rendered necessary in the constitution and regulations of the College. They were resumed in 1863, and the following members were then or subsequently added to the Committee:—

“Sir Thomas Watson, Bart., President of the College of Physicians; Mr. Luke, President of the Royal College of Surgeons; Dr. Bryson, Director-General of the Medical Department of the Navy; Dr. Balfour, Deputy Inspector-General of Hospitals and Head of the Statistical Branch at the Army Medical Board; Dr. Stark, Representative of the Registrar-General of Scotland; Dr. N. M. Burke, Representative of the Registrar-General of Ireland; Dr. Mackay, R.N., Deputy Inspector-General of Fleets; Mr. Moore, Surgeon to the Middlesex Hospitals; and Drs. C. J. B. Williams, Barlow, Arthur Farre, Black, Frederic Weber, Charles West, Chambers, Monro, George Johnson, Quain, Kirkes, Wilks, Bristowe, Henry Thomson, Hermann Weber, Gueneau de Mussy, M'William.

“A *Classification* Sub-committee was formed, consisting of—Sir Thomas Watson, Bart. (chairman), Drs. Farr, Barclay, Balfour, C. J. B. Williams, Quain, Sibson, Mr. Simon, and Mr. Holmes.

“A *Definition* Sub-committee was also formed, consisting of—Drs. Barlow, Arthur Farre, West, Chambers, Monro, George Johnson, Barclay, Sibson, Parkes, Kirkes, Wilks, Bristowe, Balfour, Mr. Moore, and Mr. Holmes.

“Mr. Gaskill and Dr. Nairne (Commissioners in Lunacy) attended the meetings of the Committee when the subject of INSANITY was under consideration.

“Mr. Cartwright and Mr. Tomes attended the meetings when the diseases of the TEETH were under consideration.

“The Latin Nomenclature was prepared by Dr. Henry Thomson, and revised by Dr. Black.

“The French Nomenclature was prepared by Dr. Gueneau de Mussy.

“The German Nomenclature was drawn up by Dr. Hermann Weber, and revised by Dr. Frederic Weber.

“The Italian Nomenclature was drawn up by Dr. Frederic Weber.

“The List of Deformities was drawn up by Dr. Arthur Farre.

“The entire work has been edited by the Secretary, Dr. Sibson; with whom Dr.

and therefore it is not unreasonably expected that greater accuracy, certainty, and uniformity, for comparison, than heretofore will characterize the statistical records of disease, alike in civil life and in the public services. To facilitate the work becoming *international*, the College has translated its nomenclature into Latin, French, German, and Italian equivalents.

The methods of gradually improving the Nomenclature of Diseases in Medicine has some analogy to the methods of gradually improving Representative Reform in Politics. A nomenclature of diseases and a policy of parliamentary representation, judicious and proper a quarter of a century ago, must each eventually give place to the influence of progressive knowledge and power, which invariably come with the rapid movements of the age in which we live. But reform, whether of political representation or of medical nomenclature, to be generally acceptable, cannot be the work of one man, nor the accomplishment of a limited period of time. To be on a broad basis and free of prejudice, each work ought to be the combined result of the best men of the time—each man being willing to yield, adapt, and mould his convictions on entering into a mutual arrangement to achieve a common end. Men thus brought together, who differ very seriously as to certain points, may yet, by mutual discussion, come to a definite and reasonable agreement for practical purposes—the terms of the compromise being settled doubtless at the expense of some personal bias, which often has a firmer hold the more imperfect the information of the holder, but which mutual

Barclay took part in editing more especially the Medical portion. The Surgical portions of the Nomenclature were prepared and, in conjunction with the Secretary, edited by Mr. Moore and Mr. Holmes.

“Official changes during the period of the existence of the Committee led also to the introduction into it of the following additional members:—Mr. Partridge, as President of the Royal College of Surgeons; Drs. Birkett, Owen Rees, Handfield Jones, Basham, Herbert Davies, Guy, Peacock, Wegg, as Censors of the College of Physicians. Dr. Alderson’s first official act after his election as President of the College was to appoint Sir Thomas Watson Chairman of the Committee.”

These names are the names of men who hold, or have held, the highest place as representative men in the Science of Medicine; and are at once a guarantee of the intellect and practical knowledge which have been brought to bear upon the work.

The preface to the Nomenclature, from the pen of the Chairman of the Committee, is extremely suggestive; and the work ought to be in the hands of every Student of Medicine, and the handbook of every one who has to do with the Registration of Diseases.

deliberation clears away. Knowing how biassed in opinion individual members of professional and political bodies are apt to be, the unanimous adoption of a Provisional Nomenclature by the London College of Physicians is undoubtedly a great and a bold achievement; and the result is a work beyond all praise.

The plan of the "Provisional Nomenclature" is, first, "*to give an English name to the disease, employing the terms in popular use whenever they are not absolutely inaccurate; and to use only one word, or as few words as possible, in NAMING a disease.*" Definitions have been attached to the English names in some instances only, as where there might be some ambiguity as to the signification which the College desires to attach to them. Thus the definitions have been framed for the purpose of identification only, not as explanations of the phenomena of disease.

The plan is, secondly, *to give a classification based upon anatomical considerations, namely,—GENERAL DISEASES, or such as affect the whole frame, subdivided into Sections A and B;* and LOCAL DISEASES.*

In studying the Science of Medicine systematically, a METHODICAL NOSOLOGY ought to be regarded as a table of reference to aid the student in naming diseases, and so preserving uniformity in his records and diagnosis, and a system to guide him generally in acquiring a knowledge of his profession, especially with reference to the practical questions of the day. The Nosology of the Royal College of Physicians of London ought, therefore, to be accepted simply as a contrivance to aid us in giving the same name to similar conditions of disease, and "for perfecting the statistical registration of diseases, with a view to the discovery of statistical truths concerning their history, nature, and phenomena."

* "Section A comprehends those disorders which appear to involve a morbid condition of the blood, and which present, for the most part, but not all of them, the following characters:—They run a definite course, are attended with fever, and frequently with eruptions on the skin, are more or less readily communicable from person to person, and possess the singular and important property of generally protecting those who suffer them from a second attack. They are apt to occur epidemically." They correspond to the *Zymotic diseases* of Dr. Farr's classification.

"Section B comprises for the most part disorders which are apt to invade different parts of the same body simultaneously or in succession. These are sometimes spoken of as constitutional diseases, and they often manifest a tendency to transmission by inheritance." They correspond to the *Constitutional diseases* of Dr. Farr's classification.

Pathology, we know, is yet too young to base a scientific classification upon; and as the Science advances, so must Nosology. But there are many nice questions which always will arise, relative to the nature of diseases, on which it is in vain to expect Physicians and Statists to agree unanimously; and therefore no system even of naming, far less of classifying, the diseases of mankind can we hope to see, otherwise than as a *provisional* one, ready to assimilate itself to the progressive advance of the Science of Medicine. We have every reason to hope that, by the numerous inquisitive researches of the day, Pathology and Nosology will grow even more rapidly than hitherto. The mere enumeration of diseases has almost doubled since Cullen's Nosology was written; while our knowledge of facts relating to disease has greatly more than doubled. Cullen's Nosology became effete and useless at last, under the pressure of increasing knowledge acquired and effected with resources very inferior to those we now possess, and far less extensive. The nomenclature and classification thus adopted by the College is therefore *strictly provisional*; and it would be well if the Colleges of Physicians and Surgeons in Scotland and in Ireland would unite with that of London in this eminently practical work, and appoint Committees to communicate with each other in revising and re-adjusting such nomenclature at the end of every ten years; and so stamp with their united authority the progressive improvements in the Science of Medicine which are capable of being indicated or expressed in the Nomenclature and Definitions of diseases. Such systematic arrangements, if consistent with existing knowledge, never cramp or hamper a man in carrying out scientific investigations; on the contrary, they enable him to see more clearly in what direction his labour must be advanced, and demonstrate more forcibly than otherwise the deficiencies of his knowledge.

The "Provisional Nomenclature" of the Royal College of Physicians of London, comprehended in the following list, has therefore been adopted in the text of this edition (5th); while the synonyms, equivalents, and definitions have also been incorporated, at the places where the diseases are described in the text, throughout both volumes.

CHAPTER II.

TABULAR VIEW OF THE "PROVISIONAL NOMENCLATURE" ADOPTED
BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

GENERAL DISEASES.

A.		
1. Small-pox. <i>Group A</i> (unmodified). <i>Group B</i> (modified). <i>Varieties</i> , applicable to both groups: <i>a.</i> Confluent. <i>b.</i> Semi-confluent. <i>c.</i> Distinct. <i>Syn.</i> , Discrete. <i>d.</i> Abortive. <i>Syn.</i> , Varicel- loid. <i>Subordinate Varieties:</i> <i>e.</i> Petechial. <i>f.</i> Hæmorrhagic. <i>g.</i> Corymbose.	15. Ague. <i>Syn.</i> , Intermittent fever. <i>Varieties:</i> <i>a.</i> Quotidian. <i>b.</i> Tertian. <i>Sub-variety:</i> Double tertian. <i>c.</i> Quartan. <i>Sub-variety:</i> Double quartan. <i>d.</i> Irregular. (100 <i>b.</i>) <i>Brow ague.</i> 16. Remittent fever.† 17. Simple cholera. 18. Malignant cholera, <i>Syn.</i> , Ser- ous cholera; Spasmodic cholera; Asiatic cholera. <i>a.</i> Choleraic diarrhœa. 19. Diphtheria. <i>a.</i> Diphtheritic paralysis. 20. Hooping-cough. 21. Mumps. 22. Influenza. 23. Glanders. 24. Farcy. 25. Equinia mitis. <i>Syn.</i> , Grease. 26. Malignant pustule. 27. Phagedæna. 28. Sloughing phagedæna. 29. Hospital gangrene. 30. Erysipelas. <i>Varieties:</i> <i>a.</i> Simple. <i>Syn.</i> , Cutaneous. <i>b.</i> Phlegmonous. <i>Syn.</i> , Cel- lulo-cutaneous. <i>c.</i> Diffuse inflammation (of cellular tissue).§ 31. Pyæmia. 32. Puerperal fever.¶	33. Puerperal ephemera. <i>Syn.</i> , Weed. B. 34. Acute rheumatism. <i>Syn.</i> , Rheumatic fever. (<i>a.</i>) Sub-acute rheumatism. 35. Gonorrhœal rheumatism. 36. Synovial rheumatism. 37. Muscular rheumatism. <i>Local varieties:</i> <i>a.</i> Lumbago. <i>b.</i> Stiff neck. 38. Chronic rheumatism. ** 39. Acute gout. 40. Chronic gout. 41. Gouty synovitis.†† 42. Chronic osteo-arthritis. <i>Syn.</i> , Chronic rheumatic arthritis. 43. Purpura. <i>Varieties:</i> <i>a.</i> Simple. <i>b.</i> Hæmorrhagic. 44. Scurvy. <i>Ergotism.</i> 45. Diabetes. <i>Syn.</i> , Diabetes mel- [itus]. 46. Syphilis. [litus]. <i>a.</i> Primary syphilis. <i>Varieties:</i> Hard chancre. Indurated bubo. Soft chancre. Suppurating bubo. Phagedænic sore. Sloughing sore. <i>b.</i> Secondary syphilis. <i>c.</i> Hereditary syphilis. 1. †† Local syphilitic affections.

* Scarlet fever occurs occasionally without any rash or sore-throat being observed.

† Fevers symptomatic of worms, teething, or other sources of irritation, should not be included under this head.

‡ The malignant local fevers of warm climates are usually of this class.

§ In slighter cases, occurring on the surface of the body, this disease is identical with phlegmonous erysipelas.—In registering cases of phlegmonous erysipelas, and of diffuse inflammation arising from injury, surgical operation, or local disease, the cause should be specified.

|| In returning cases of pyæmia, specify the affected organs.

¶ In returning cases of puerperal fever, the more important local lesions, such as peritonitis, effusions into serous and synovial cavities, phlebitis, and diffuse suppuration, should be specified.

** Cases attended with deposit of lithate of soda are to be returned as chronic gout, and those in which there is marked distortion as chronic osteo-arthritis.

†† Retrocedent gout is a term applied to cases of gout in which some internal organ becomes affected in the disappearance of the disease from the joints, and should be referred to acute or chronic gout.

‡‡ In returning local syphilitic affections, specify whether the case be one of primary syphilis, second-ary syphilis, syphilitic deposit, or syphilitic inflammation.—Local syphilitic affections, local cancer, local oloid, and local scrofulous affections are to be returned in the following order:—

Brain.	Supra-renal capsule.	Stomach.	Scrotum.
Spinal cord.	Larynx.	Pylorus.	Testicle.
Nerve.	Bronchi.	Intestines.	Ovary.
Eye.	Lungs.	Rectum.	Fallopian tube.
Eyelid.	Pleura.	Anus.	Uterus.
Orbit.	Mediastinum.	Liver.	Vagina.
Auricle.	Lips.	Hepatic ducts and gall- bladder.	Vulva.
Internal ear.	Mouth.	Pancreas.	Female breast.
Face.	Cheek.	Spleen.	Male mamilla.
Nose.	Jaws.	Peritoneum.	Bone.
Pericardium.	Gum.	Mesenteric glands.	Skull.
Heart.	Tongue.	Kidney.	Joint.
Lymphatics.	Fauces.	Bladder.	Spine.
Lymphatic glands.	Tonsils.	Prostate gland.	Muscle.
Bronchial glands.	Salivary glands.	Penis.	Tendon.
Thyroid gland.	Pharynx.	Prepuce.	Cellular tissue.
Thymus gland.	Œsophagus.		Skin.

47. Cancer. *Syn.*, Malignant disease.**Varieties:*

- [*cer.*
a. Scirrhus. *Syn.*, Hard cancer.
b. Medullary cancer. *Syn.*, Soft cancer.
c. Epithelial cancer. *Syn.*, Canceroid epithelioma.
d. Melanotic cancer. *Syn.*, Melanosis.
e. Osteoid cancer.†

1. Local cancer.‡

48. Colloid. *Syn.*, Colloid cancer; Alveolar cancer.

1. Local colloid.§

NON-MALIGNANT TUMORS AND CYSTS.||

- Fibrous tumor.
 Fibro-cellular tumor.¶
 Fibro-nucleated tumor.
 Fibro-plastic tumor.**
 Myeloid tumor.
 Fatty tumor. *Syn.*, Lipoma.
 Osseous tumor.
a. Of bone. *Syn.*, Exostosis.
Varieties: Ivory.
 Cancelled.
 Diffused.
b. Of the soft parts.
 Cartilaginous tumor. *Syn.*, Enchondroma.

Fibro-cartilaginous tumor.
 Glandular tumor. *Syn.*, Adenocoele.

- Vascular tumor.
 Nævus.
 Sebaceous tumor.
 Cholesteotoma.
 Molluscum.
 Warty tumor and warts.
 Condyloma.
 Cheloid.
 Villous tumor.
 Simple or barren cysts.

- a.* Serous.
b. Synovial. *Syn.*, Bursal.
c. Mucous.
d. Suppurating.
e. Sanguineous.
f. Hæmorrhagic.
g. Aneurismal.
h. Oily.
i. Colloid or gelatinous.
j. Seminal.

Compound or proliferous cysts.

- a.* Complex cystic tumor. *Syn.*, Cysto-sarcoma. With intracystic growths.
b. Cutaneous or piliferous. *Syn.*, Dermoid.
c. Dentigerous.

49. Lupus.

Varieties:

- a.* Chronic lupus.
b. Lupus exedens.

50. Rodent ulcer.

51. True leprosy. *Syn.*, Elephantiasis Græcorum.

52. Scrofula.

Varieties:

- a.* Scrofula with tubercle.
b. Scrofula without tubercle.††

1. Local scrofulous affections.

- Tubercular meningitis.
 Scrofulous ophthalmia.
 Scrofulous iritis.
 Tubercular pericarditis.
 Phthisis pulmonalis.
 Scrofulous disease of glands.
 Acute miliary tuberculosis.
 Tabes mesenterica.
 Tubercular peritonitis.††

53. Rickets.

54. Cretinism.

Varieties:

- a.* Complete cretinism. *Syn.*, Incurable cretinism.
b. Demi-cretinism.
c. Incomplete cretinism. *Syn.*, Curable cretinism.

55. §§ Anæmia.

56. Chlorosis. *Syn.*, Green-sickness.

57. §§ General dropsy. |||

DISEASES OF THE NERVOUS SYSTEM.¶¶

DISEASES OF THE BRAIN AND ITS MEMBRANES.

58. Encephalitis.***

59. Meningitis.

1. Inflammation of the dura mater.†††

2. Inflammation of the pia mater and arachnoid.
 (59') 3. Tubercular meningitis. *Syn.*, Acute hydrocephalus.
 60. Inflammation of the brain.
 61. Red softening (of the brain).
 62. Yellow softening (of the brain).

63. Abscess (of the brain).

64. Apoplexy.

Varieties:

- a.* Congestive.
b. Sanguineous. *Syn.*, Cerebral hæmorrhage.

65. Sunstroke.

* In returning cases of cancer in more than one organ, specify in which the disease is primary, and in which secondary.—State also the kind and duration of the disease in each case, and the nature of all operations, with their dates and results.

† Cancer in mucous membranes, when covered by a villous growth, has received the name of Villous cancer.

‡ In returning cases of local cancer, specify the variety of cancer, by adding, after "46," the letter *a*, *b*, *c*, *d*, or *e*, according to the nature of the case. They are to be returned in the order specified in the foot-note (††) on preceding page.

§ Cases of local colloid are to be returned in the order specified in the foot-note (††) on preceding page.

|| In order that the malignant and non-malignant growths may appear together, the non-malignant tumors and cysts are inserted here. They should, however, be returned under "Non-malignant tumors" among the local diseases, and they are not, therefore, numbered at this place.

¶ When occurring as a pendulous outgrowth from a mucous surface, it constitutes the chief varieties of Polypus.

** When the fibro-cellular or fibro-plastic tumor, but more especially the latter, slowly involves the adjacent soft structures, and returns after removal, it has received the name of Recurrent fibroid.

†† The constitutional tendency which has received the name of the Scrofulous Diathesis, when unattended by local lesions, is not to be returned under the heading of Scrofula.

‡‡ These and all other cases of local scrofulous affection are to be returned in the order specified in the foot-note (††) on preceding page.

§§ When the cause of this affection has been ascertained, the case should be returned under the heading of the Primary disease, the secondary affection being also specified.

||| Local dropsies, such as ovarian, and effusions into the serous cavities, as hydrothorax or ascites, when not connected with anasarca, should be returned as local diseases.

¶¶ ARRANGEMENT OF LOCAL DISEASES.

The Local Diseases have been drawn up in accordance with the following arrangement:—

Catarrh.	Gangrene.	Atrophy.	Cyst.
Inflammation.	Passive congestion.	Degeneration.	Scrofula.
Ulcerative inflammation.	Extravasation of blood.	Fatty and Calcareous.	<i>a.</i> With tubercle.
Suppurative	Hæmorrhage.	<i>Syn.</i> , Atheromatous.	<i>b.</i> Without tubercle.
Plastic	Dropsy.	Fibroid.	Parasitic disease.
<i>Pyæmic</i>	Fibrinous deposit.	Lardaceous disease.	Calculus and concretion.
Rheumatic	Alteration of dimensions.	<i>Syphilitic disease.</i>	Malformation.
Gouty	Dilatation.	Cancer.	Injury.
<i>Syphilitic</i>	Contraction.	Colloid.	Foreign body.
Scrofulous	Hypertrophy.	Non-malignant tumor.	Functional diseases.
Gonorrhœal			

The diseases printed in italics are to be returned, not among the local diseases, but under the heading referred to by number.

*** This term is to be used only when the precise seat of the inflammation has not been ascertained by *post-mortem* examination.

††† This form of inflammation is almost invariably the result of injury or disease of the bones of the skull; in such cases, the injury or disease by which it is caused ought to be specified.

66. Chronic hydrocephalus.
 67. Hypertrophy (of the brain).
 68. Atrophy (of the brain).
 69. White softening (of the brain).
Syn., Atrophic softening.*
 (481.) *Syphilitic disease.*
 (491.) *Cancer.*
 70. Fibrous tumor
 71. Osseous tumor.
 (531.) *Tubercular deposit.*
a. Miliary or granular tubercle.†
b. Yellow tubercle.
 72. Hydatid.
 73. Diseases of the cerebral arteries.
 1. Degeneration (fatty and calcareous). *Syn.*, Atheroma. Ossification.
 2. Aneurism.
 3. Impaction of coagula.
 1. Thrombosis (local coagulation).
 2. Embolism (coagula conveyed to a distance).

DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

74. Inflammation.‡
Varieties:
a. Spinal meningitis.
b. Myelitis.
 75. Hæmorrhage (spinal). *Syn.*, Spinal apoplexy.

76. Atrophy (spinal). *Syn.*, Tabes dorsalis.
 77. White softening (spinal).
 78. Spina bifida.
 (491.) *Cancer.*
 79. Non-malignant tumor.

DISEASES OF NERVES.

80. Inflammation.
 81. Atrophy.
 (49.) *Cancer.*
 82. Neuroma.
 83. § Paralysis.
 (107.) 1. *Paralysis of the insane.*
Syn., General paralysis.
 84. 2. § Hemiplegia.
 85. 3. § Paraplegia.
 86. 4. Locomotor ataxy.
 (791.) *Progressive muscular atrophy.*
 87. 5. Infantile paralysis.
 6. § Local paralysis.¶
a. Scrivener's palsy.
 7. *Diphtheritic paralysis.*
 8. *Lead palsy.*

GENERAL DISEASES OF THE NERVOUS SYSTEM.

88. Tetanus.
 89. Hydrophobia.
 90. Infantile convulsions.
 91. Epilepsy.
 Epileptic vertigo. *Syn.*, Petit mal.
 92. § Convulsions.
 93. Spasm of muscle.

94. Laryngismus stridulus. *Syn.*, Spasm of the glottis. Spasmodic croup. Child-crowing.
 95. Shaking palsy.
 96. *Mercurial tremor.*
 97. Chorea. *Syn.*, St. Vitus's dance.
a. Acute.
b. Chronic.
 98. Hysteria.
 99. Catalepsy.
 100. Neuralgia.
Principal varieties:
a. Facial. *Syn.*, Tic douloureux.
b. Brow ague. *Syn.*, Hemicrania.
c. Sciatica.
 101. § Hyperæsthesia.
 102. § Anæsthesia.
 103. *Delirium tremens.*
 110. Hypochondriasis.

DISORDERS OF THE INTELLECT, &c.

104. Mania.
a. Acute mania.
b. Chronic mania.
 105. Melancholia.¶
 106. Dementia.
a. Acute dementia.
b. Chronic dementia.
 107. Paralysis of the insane. *Syn.*, General paralysis.
 108. Idiotey. (Congenital.)
 109. Imbecility. (Congenital.)

DISEASES AND INJURIES OF THE EYE.

CONJUNCTIVA.

111. Conjunctivitis. *Syn.*, Ophthalmia.
 112. Catarrhal ophthalmia.
 113. Pastular ophthalmia.
 114. Purulent ophthalmia.
 115. Purulent ophthalmia of infants. *Syn.*, Ophthalmia neonatorum.
 (531.) *Scrofulous ophthalmia.* *Syn.*, *Strumous ophthalmia.*
 116. Exanthematous ophthalmia.
 117. Gonorrhœal ophthalmia.
 118. Chronic ophthalmia.
 119. Oedema of the sub-conjunctival tissue. *Syn.*, Chemosis.
 120. Pinguecula.
 121. Pterygium.
 122. Fatty tumors.
 123. Cysticercus telæ cellulosa.
 124. Metallic stains.
a. From nitrate of silver.
b. From lead.

CORNEA.

125. Keratitis.
 126. Chronic interstitial keratitis.
 127. Keratitis with suppuration. *Syn.*, Onyx.
 128. Ulcer.
 129. Opacity. *Syn.*, Leucoma.
 130. Conical cornea.
 131. Arcus senilis.
 132. Staphyloma.
 133. Entozoa in the anterior chamber.

SCLEROTIC.

134. Sclerotitis.

135. Staphyloma.

IRIS.

136. Iritis.
 137. Traumatic iritis.
 138. Rheumatic iritis.
 139. Arthritic iritis.
 (401.) *Syphilitic iritis.*
 (531.) *Scrofulous iritis.*
 140. Gonorrhœal iritis.
 141. Sequelæ of iritis.
 142. Congenital defects of iris.

CHOROID AND RETINA.

143. Choroiditis.
 144. Retinitis.
 145. Choroidal apoplexy.
 146. Amaurosis.
 147. Impaired vision.
 148. Muscæ volitantes.
 149. Albinism.

VITREOUS BODY.

150. Synchysis.
 151. Various morbid deposits.
 152. Entozoa.

LENS AND ITS CAPSULE.

153. Cataract.
Varieties:
a. Hard.
b. Soft.
c. Fluid.
 154. Congenital cataract.
 155. Traumatic cataract.

GENERAL AFFECTIONS OF THE EYE.

156. Glaucoma.

157. Hydrophthalmia.
 (491.) *Cancer.*
 (53.) *Scrofulous deposit within the eyeball.*
 158. Total disorganisation of the eye from injury.

VARIOUS DEFECTS OF SIGHT.

159. Short sight.
 160. Long sight.
 161. Faulty perception of colours. *Syn.*, Colour blindness.
 162. Hemeralopia.
 163. Nyctalopia.
 164. Astigmatism.

DISEASES OF THE LACHRYMAL APPARATUS.

165. Lachrymal obstruction.
 166. Abscess and fistula.
 167. Dacryolith.
 168. Diseases of the lachrymal gland and its ducts.

DISEASES OF THE EYELIDS.

169. Inflammation.
 170. Hordeolum.
 171. Abscess in the Meibomian glands.
 172. Epicanthis.
 173. Entropium.
 174. Ectropium.
 175. Trichiasis.
 176. Madarosis. *Syn.*, Loss of the eyelashes.
 177. Tarsal ophthalmia.
 178. Blepharospasmus.
 (49.) *Cancer.*

* This form of disease is the result of imperfect nutrition, owing to deficient supply of blood, and is in most instances dependent upon mechanical obstruction, or degeneration of the cerebral arteries.

† To be referred to tubercular meningitis.

‡ This term is to be used only when the precise seat of the inflammation has not been ascertained by post-mortem examination.

§ When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

¶ When the cause of any of these forms of paralysis has been ascertained, it should be stated.

¶ Cases of so-called monomania to be classed under chronic mania or melancholia, according to their character.

179. Cyst of the lids.
Phthiriasis.

DISEASES OF THE PARTS WITHIN
THE ORBITS.

180. Abscess in the orbit.

AURICLE.

186. Gouty and other deposits.
187. Hæmatoma auris.
(491.) *Cancers.*
188. Non-malignant tumor.
(821. &c.) *Cutaneous affections.*
189. Malformations.
(1007.) *Injuries.*

EXTERNAL MEATUS.

190. Inflammation.
 a. Acute.
 b. Chronic.
191. Abscess.
192. Accumulation of wax.

181. Strabismus.
182. Protrusion of the eyeball.
 Syn., Proptosis.
(277.) *Ex-ophthalmic bronchocele.*
(248.) *Orbital aneurism.*
(49.) *Cancer.*

DISEASES OF THE EAR.

193. Polypus.
194. Sebaceous tumor. *Syn.,* Mol-
 luscous.
195. Exostosis.
(1007.) *Foreign bodies.*

MEMBRANA TYMPANI.

196. Inflammation.
197. Ulceration.
198. Perforation.
(1007.) *Injuries.*

EUSTACHIAN TUBE.

199. Obstruction.

TYMPANUM.

200. Disease of the mucous mem-
 brane.
201. Disease of the ossicles.
202. " " mastoid cells.

INTERNAL EAR.

203. Organic disease.
204. Necrosis of petrous bone.
205. Deafness.
 a. Functional or nervous.
 b. From disease.
 c. Deaf-dumbness.
(491.) *Cancer.**

DISEASES OF THE NOSE.

206. Hypertrophy. *Syn.,* Lipoma.
207. Wart.
208. Sebaceous cyst.
(491.) *Cancer of the skin.*
(511.) *Lupus.*
209. Ozena.
210. Abscess of the septum.
210. Ulceration of the pituitary
 membrane.

211. Perforation of the septum.
212. Epistaxis.
213. Hypertrophy of the pituitary
 membrane.
214. Polypus nasi.
 Varieties:
 a. Gelatinous.
 b. Fibrous.
 Naso-pharyngeal polypus.

215. Non-malignant tumors of the
 septum.
216. Rhinoliths.
(1008.) *Foreign bodies.*
(491.) *Cancer. Syn., Malignant poly-
 pus.*
 Loss or perversion of sense
 of smell.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE HEART AND ITS
MEMBRANES.

- DISEASES OF THE PERICARDIUM.
216. Pericarditis.
217. Suppurative pericarditis.
(531.) *Tubercular pericarditis.*
218. Adherent pericardium.†
219. Dropsy.
(491.) *Cancer.*
220. Malformations.

DISEASES OF THE ENDOCARDIUM.

221. Endocarditis.‡
222. Valve-disease.
 1. Aortic.
 2. Mitral.
 3. Of pulmonary artery.
 4. Tricuspid.
 Varieties:
 a. Vegetations.
 b. Fibroid thickening.
 c. Atheromatous and cal-
 careous degeneration.
 d. Aneurism.
 e. Laceration.
 f. Simple dilatation of ori-
 fice.
 g. Malformations.
 Obstruction to the cir-
 culation, or
 Regurgitations should be
 specially noted when
 they accompany the
 valve disease.

DISEASES OF THE MUSCULAR STRUC-
TURE OF THE HEART.

223. Myocarditis.
224. Abscess.¶
225. Hypertrophy.
 a. Of left side.
 b. Of right side.
226. Dilatation.
 a. Of left side.
 b. Of right side.
227. Atrophy.
228. Excess of fat.
229. Fatty degeneration.
230. Fibroid degeneration.
231. Aneurism.
232. Acute aneurism.
233. Rupture.¶
(491.) *Cancer.*
234. Entozoa.
235. Disease of the coronary
 arteries.
236. Malformations.**
237. Cyanosis.
238. *Injuries of the heart.*
239. †† Angina pectoris.
240. †† Syncope.
241. †† Palpitation and irregularity
 of the heart.

DISEASES OF THE BLOOD-VESSELS.††

DISEASES OF THE ARTERIES.

242. Arteritis.

243. Degeneration. Fatty and
 calcareous. *Syn.,* Atheroma.
 Ossification.
244. Narrowing and obliteration.
246. Occlusion.
 a. From compression.
 b. From impaction.
 1. Thrombosis (local coa-
 gulation).
 2. Embolism (coagula con-
 veyed from a distance).
247. Dilatation.
248. Aneurism.
 In returning such cases, state
 whether the aneurism be—
 a. Fusiform,
 b. Saccular, or
 c. Diffused (sac formed by
 the surrounding tissues).
249. Rupture of artery.
 a. From disease of artery.
 b. From disease external
 to artery.
250. Partial rupture of artery.
 Syn., Dissecting aneurism.
251. Traumatic.
252. Arterio-venous aneurism.
253. Aneurismal varix.
 Varieties:
 a. Traumatic.
 b. Spontaneous.
254. Varicose aneurism.
 Varieties:
 a. Traumatic.
 b. Spontaneous.

* When any of these affections implicate the brain, carotid artery, or lateral sinus, the fact should be stated.

† Including under this term partial adhesions and calcareous and ossific deposits.

‡ In returning such cases, state, if possible, the valve or valves affected.

§ Cases are to be returned under this head only when the condition has evidently existed during life, and is believed to have been the cause of death.

¶ Abscess dependent on pyæmia should be referred to that disease.

¶ In returning cases of aneurism and rupture, the situation ought to be stated.

** State which, according to list, page 198.*

†† When the cause of this affection has been ascertained, the case should be returned under the head of the Primary disease, the secondary affection being also specified.

‡‡ The vessel affected should in all cases be specified.

. Cirroid aneurism. <i>Syn.</i> , Arterial varix. . Aneurism by anastomosis. . Malformation. <i>a.</i> Constriction or occlusion of the commencement of the descending aorta (originating in partial malformation). (99 <i>a.</i>) <i>Injuries of arteries.</i> (99 <i>a1.</i>) <i>Contusion.</i>	(104 <i>a2.</i>) <i>Laceration. Syn., Partial</i> <i>rupture.</i> <i>a.</i> Of outer coat. <i>b.</i> Of inner coat. <i>Wound.</i> DISEASES OF VEINS. 257. Phlebitis. <i>Varieties:</i> <i>a.</i> Adhesive. Phlegmasia dolens. <i>b.</i> Suppurative.	258. Phlegmasia dolens. 258. Fibrinous concretions in the 259. Obstruction. [veins. 260. Obliteration. 261. Phlebolithes. 262. Varicose veins. 263. Nævus vascularis. (104 <i>b.</i>) <i>Injuries of veins.</i> (104 <i>b1.</i>) <i>Rupture, without exter-</i> <i>nal wound.</i> (1049' 2.) <i>Wound of vein, with en-</i> <i>trance of air.</i>
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DISEASES OF THE ABSORBENT SYSTEM.

Inflammation of lymphatics. Suppuration of lymphatics. Inflammation of glands. Suppuration of glands. Hypertrophy of glands. <i>a.</i> Chronic enlargement of glands. Atrophy of glands. Lymphatic fistula. 2.) Foreign bodies and concre- tions.	271. Obstruction of the thoracic duct.* 272. Obstruction, obliteration, and varicosity of lymphatics. 273. Bursting of lymphatics. (49.) <i>Syphilitic bubo.</i> (49.) <i>Syphilitic inflammation of</i> <i>glands.</i> (50.) <i>Cancer.</i> (53.) <i>Scrofulous disease of glands.</i> <i>Suppuration.</i>	(1133.) <i>Wounds of lymphatics.</i> <i>Diseases of the Bronchial Glands.</i> (338.) <i>Inflammation.</i> (339.) <i>Abscess.</i> (340.) <i>Enlargement.</i> (49.) <i>Cancer.</i> (341.) <i>Non-malignant growth.</i> (531.) <i>Tubercle.</i>
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DISEASES OF DUCTLESS GLANDS.

DISEASES OF THE THYROID GLAND. Inflammation. <i>a.</i> Acute. <i>b.</i> Chronic. Goitre. Cyst. Exophthalmic bronchocele. Pulsating bronchocele.	(491.) <i>Cancer.</i> DISEASES OF THE THYMUS GLAND. 279. Hypertrophy. (50.) <i>Cancer.</i> 280. Non-malignant tumor.	DISEASES OF THE SUPRA-RENAL CAPSULES. 281. Suppuration. (491.) <i>Cancer.</i> (53.) <i>Tubercular degeneration.</i> 282. Addison's disease. <i>Syn.</i> , Bronze skin. Melasma Addisoni.
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DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE RESPIRATORY SYS- TEM NOT STRICTLY LOCAL. Hay asthma. Influenza. Hooping cough. Croup. Diphtheria.) † <i>Asphyxia.</i> DISEASES OF THE NOSTRILS. Coryza. <i>Syn.</i> , Nasal catarrh. DISEASES OF THE LARYNX. Inflammation of the epiglottis. Ulceration of the epiglottis. Laryngeal catarrh. Laryngitis. <i>a.</i> Acute. <i>b.</i> Chronic. Ulcer. ‡ Abscess. Edema of the glottis. Necrosis of cartilage (see the previous Note). Contraction. Epithelial cancer. Warty growth. Polypus. Cyst.) Foreign bodies in the larynx. FUNCTIONAL AFFECTIONS OF THE LARYNX. Aphonia. Paralysis of the glottis. Spasm of the glottis. Laryngismus stridulus.	DISEASES OF THE TRACHEA AND BRONCHI. 305. Bronchial catarrh. 306. Bronchitis. <i>a.</i> Acute. <i>b.</i> Chronic. 307. Ulcer. 308. † Casts of the bronchial tubes. 309. Necrosis of the cartilages of the trachea. § 310. Dilatation. 311. Contraction. (491.) <i>Cancer.</i> 312. Non-malignant tumor. (531.) <i>Tubercle.</i> (1036.) <i>Foreign body in the bronchi.</i> 313. Asthma. DISEASES OF THE LUNG. 314. Pneumonia. <i>Variety:</i> <i>a.</i> Lobular. 315. Abscess. (28.) <i>Pyæmic inflammation and ab-</i> <i>scess.</i> 316. Gangrene. 317. † Passive congestion. 318. † Pulmonary extravasation. Pulmonary apoplexy. 319. † Edema. 320. Cirrhosis. 321. Emphysema. <i>a.</i> Vesicular. † <i>b.</i> Interlobular. 322. Atelectasis. 323. † Collapse. (481.) <i>Syphilitic deposit.</i>	(491.) <i>Cancer.</i> (531.) <i>Phthisis.</i> (531.) <i>Acute miliary tuberculosis.</i> 324. Acute pneumonic phthisis. 325. Chronic pneumonic phthisis. 325. Hydatid. (1042-46.) <i>Injuries.</i> (1036.) <i>Foreign bodies.</i> 326. Millstone makers' phthisis. 327. Grinders' asthma. 328. Miners' Asthma. DISEASES OF THE PLEURA. 329. Pleurisy. 330. Chronic pleurisy. 331. Empyema. 332. Adhesions, including thicken- ing and ossification. 333. † Hydrothorax. 334. Pneumothorax. (491.) <i>Cancer.</i> 335. Non-malignant tumor. (531.) <i>Tubercular pleurisy.</i> (1045.) <i>Injuries.</i> DISEASES OF THE MEDIASTINUM. 336. Abscess. (491.) <i>Cancer.</i> 337. Non-malignant tumor. (279.) <i>Diseases of the thymus gland.</i> DISEASES OF THE BRONCHIAL GLANDS. 338. Inflammation. 339. Abscess. 340. Enlargement. (491.) <i>Cancer.</i> 341. Non-malignant tumor. (531.) <i>Tubercle.</i>
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The cause of the obstruction should be stated.
 When the cause of this affection has been ascertained, the case should be returned under the head of primary disease, the secondary affection being also specified.
 When chronic laryngitis, ulcer of the larynx, or necrosis of cartilage (see below), is due to phthisis or to syphilis, the terms *Syphilitic* or *Phthisical* should be prefixed to the designation of the disease, and the case to be returned under the head of the primary affection.
 When this affection is due to phthisis or syphilis, the terms *Syphilitic* or *Phthisical* should be prefixed to the designation of the disease, and the case ought to be returned under the head of the primary affection.
 The term Secondary has been applied to pneumonia when it occurs as a complication of some other disease; such cases ought to be returned under the head of the primary affection.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE LIPS.

The affected lip ought to be specified.

- 342. Ulcer.
- (481.) *Syphilitic ulcer.*
- 343. Fissures.
- (491.) *Cancer.*
- (531.) *Strumous hypertrophy.*
- 344. Cyst.
- 345. Malformations.
- 346. Hare-lip.

DISEASES OF THE MOUTH.

- 347. Stomatitis.
- 348. Ulcerative stomatitis.
- 349. Thrush. *Syn.*, Aphtha. Vesicular stomatitis.
- 350. Parasitic thrush. *Syn.*, Parasitic aphthæ.
- 351. Abscess of the cheek.
- 352. Cancrum oris. *Syn.*, Gangrenous stomatitis.
- 353. Cysts of the cheek.
- 354. Ranula.
- (491.) *Cancer.*

DISEASES OF THE JAWS (exclusive of the Alveoli).

- 355. Adhesion of the jaws by cicatrix.
- 353. Abscess of the antrum.
- (491.) *Cancer.*
- 357. Fibrous tumor.
- 358. Myeloid tumor.
- 359. Osseous tumor.
- Hypertrophy of the bones of the face.
- 360. Cartilaginous tumor.
- 361. Vascular tumor.
- 362. Cyst.

DISEASES, MALFORMATIONS, AND INJURIES OF THE TEETH, GUMS, AND ALVEOLI.

- 363. Teething.*

DISEASES OF THE DENTAL TISSUE.

- 364. Caries.
- 365. Necrosis.
- 366. Exostosis.
- 367. Absorption.

DISEASES OF THE DENTAL PULP.

- 368. Irritation
- 369. Inflammation.
- 370. Ulceration.
- 371. Gangrene.
- 372. Granulation or polypus.
- 373. Calcification.

DISEASES OF THE DENTAL PERIOSTEUM.

- 374. Inflammation.
- 375. Gum-boil.
- 376. Chronic thickening.
- 377. Rheumatic inflammation.

DISEASES OF THE GUMS.

- 378. Inflammation.
- 379. Ulceration.
- 380. Hypertrophy.
- 381. Atrophy.
- 382. Induration (in infancy).
- (501.) *Cancer.*
- 383. Non-malignant tumor.
- a. Polypus.
- b. Cartilaginous tumor.
- c. Vascular tumor.
- 384. Epulis.

DISEASES OF THE ALVEOLI.

- 385. Inflammation.
- 386. Necrosis.
- 387. Caries.
- 388. Exostosis.
- 389. Dentigerous cyst.
- 390. Absorption.

SPECIFIC DISEASES AFFECTING THE DENTAL PERIOSTEUM, GUMS, OR ALVEOLI.

- 391. Mercurial inflammation.
- 392. Phosphoric inflammation and necrosis.
- (902.) *Blue gum from lead.*
- (45.) *Scurvy.*

IRREGULAR DENTITION.

- Irregularity in the time of eruption of the—
- 393. Temporary teeth.
- 394. Permanent teeth.
- Irregularity in the position of the—
- 395. Temporary teeth.
- 396. Permanent teeth.
- Irregularity of the number of the—
- 397. Temporary teeth.
- 398. Permanent teeth.
- Irregularity in the form of the—
- 399. Temporary teeth.
- 400. Permanent teeth.
- Abnormal development of the—
- 401. Dental tissue.
- 402. Enamel.
- 403. Dentine.
- 404. Cementum.
- 405. Alveolar portions of the jaws, in size.
- 406. Alveolar portions of the jaws, in form.
- 407. Defective growth of lower jaw.
- 408. Mechanical injuries of the alveoli and dental periosteum.
- a. Hæmorrhage.
- b. Fracture.
- 409. Mechanical injuries of the teeth.
- a. Fracture.
- b. Dilaceration.
- c. Dislocation.
- d. Friction.

DISEASES OF THE TONGUE.

- 410. Glossitis.
- 411. Ulceration.
- 412. Aphthous ulcer.
- 413. Abscess.
- 414. Hypertrophy.
- (481.) *Primary syphilis.*
- (481.) *Secondary syphilis.*
- (49.) *Epithelial cancer.*
- 415. Vascular tumor.
- 416. Tongue-tie.
- (87.) † *Paralysis.*

DISEASES OF THE FAUCES AND PALATE.

- 417. Sore throat.
- 418. Relaxed throat.
- 419. Ulcerated throat.
- 420. Quinsy. *Syn.*, Cynanche tonsillar.
- 421. Tonsillitis.
- 422. Sloughing sore throat. *Syn.*, Putrid sore throat. Cynanche maligna. †
- (19.) *Diphtheria.*

- 423. Enlarged tonsils.
- (491.) *Cancer of tonsils.*
- (531.) *Scrofulous diseases of tonsils.*
- 424. Elongated uvula.
- 425. Perforation of the palate.
- 426. Stricture of the fauces.
- (481.) *Syphilitic affection of fauces and tonsils.*
- (491.) *Cancer.*
- 427. Non-malignant tumor.
- a. Fibro-cellular tumor.
- b. Fibro-cystic tumor.
- 428. Cleft palate.

DISEASES OF THE PHARYNX.

- 429. Pharyngitis.
- 430. Ulcer.
- a. Superficial ulcer.
- b. Perforating ulcer.
- 431. Abscess.
- 432. Sloughing.
- 433. Adhesion of soft palate.
- 434. † Dilatation.
- (481.) *Syphilitic affection.*
- (491.) *Cancer.*
- (1037.) *Foreign bodies.*
- (87.) † *Paralysis.*

DISEASES OF THE SALIVARY GLANDS.

- 435. Inflammation.
- 436. † Salivation. *Syn.*, Ptyalism.
- 437. Abscess.
- 438. Salivary fistula.
- (21.) *Mumps.*
- (491.) *Cancer.*
- 439. Non-malignant tumor.
- 440. Salivary calculus. §

DISEASES OF THE ŒSOPHAGUS.

- 441. Œsophagitis.
- 442. Ulceration.
- 443. † Perforation.
- 444. † Stricture.
- (491.) *Cancer.*
- (1038.) *Foreign bodies.*
- 445. Malformations.
- (83.) † *Paralysis.*
- 446. Dysphagia.

DISEASES OF THE STOMACH.

- 447. Gastritis.
- (916, &c.) a. *From irritant poison.*
- 448. Chronic ulcer.
- 449. Hæmatemesis.
- 450. Perforation. ||
- 451. † Dilatation.
- 452. † Stricture.
- 453. Gastric fistula.
- 454. Hernia.
- (491.) *Cancer.*
- (501.) *Colloid.*
- 455. Non-malignant tumor.
- 456. Sarcinæ.
- (1061-63.) *Injuries to the stomach.*
- (1065.) *Foreign bodies.*
- 457. Laceration (spontaneous).
- 458. Dyspepsia.
- 459. Gastrodynia.
- 460. Pyrosis.
- 461. † Vomiting.

DISEASES OF THE INTESTINES.

- 462. Enteritis.
- 463. Typhlitis.
- 464. Dysentery.
- 465. Ulceration.
- 466. Perforation.

* Any affection, such as convulsions and paralysis, induced by this condition, should be specified.

† When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

‡ This affection must be distinguished from malignant scarlet fever.

§ Whenever any of the affections of the mouth, throat, or parts connected therewith depend on syphilis, scurvy, local irritants, or any other specific cause, the fact should be stated.

|| The cause of the perforation, when ascertained, should be stated.

67. Abscess in the sub-peritoneal tissue.
 68. Fæcal abscess.
 69. Fistula.
 a. Fæcal fistula. *Syn.*, Artificial anus.
 557.) Vesico-intestinal fistula.
 70. Hæmorrhage.
 71. Melæna.
 72. * Dilatation.
 73. * Tympanites.
 74. * Obstruction.
 75. Stricture.
 76. Intussusception.
 77. Internal strangulation.
 a. Mesenteric.
 b. Mesocolic.
 78. Hernia.
 a. Reducible.
 b. Irreducible.
 c. Obstructed.
 d. Inflamed.
 e. Strangulated.
 1. Diaphragmatic.
 2. Epigastric.
 3. Ventral.
 4. Umbilical.
 5. Lumbar.
 6. Inguinal.
 a. Oblique.
 b. Direct.
 c. Incomplete.
 d. Scrotal.
 e. Congenital.
 7. Femoral.
 8. Obturator.
 9. Perineal.
 10. Pudendal.
 11. Vaginal.
 12. Ischiatic.
 479. Diseases of hernial sacs.
 a. Inflammation.
 b. Fibrinous effusion with closure.
 c. Suppuration.
 d. Dropsy.
 e. Movable bodies.
 f. Laceration.
 (491.) *Cancer.*
 (501.) *Colloid.*
 480. Non-malignant tumor.
 a. Polypus.
 481. Worms.
 (1066.) *Concretions.*
 (1066.) *Foreign bodies.*
 (1061-63.) *Injuries.*
 482. Diarrhœa.
 (17.) *Simple cholera.*

- (18.) *Malignant cholera.*
 a. *Choleraic diarrhœa.*
 483. * Paralysis.
 484. Colic.
 (902^a.) *Lead colic.*
 485. Constipation.

DISEASES OF THE RECTUM AND ANUS.

486. Ulcer.
 487. Abscess.
 488. Fistula in ano.
 (558.) *Recto-vesical fistula.*
 (586.) *Recto-urethral fistula.*
 (670.) *Recto-vaginal fistula.*
 489. Hæmorrhoids.
 a. Internal.
 b. External.
 490. Hæmorrhage from rectum.
 491. Fissure of the anus.
 492. Prolapsus.
 493. * Stricture.
 (49.) *Cancer of the rectum.*
 (49.) *Cancer of the anus.*
 Syphilis of rectum.
 Condyloma of anus.
 494. Non-malignant tumor of the rectum.
 a. Polypus.
 (1072, 73.) *Injuries.*
 (1079.) *Foreign bodies.*
 495. Neuralgia.
 496. Spasm of the sphincter ani.
 497. Pruritus ani.

DISEASES OF THE LIVER.

498. Hepatitis.
 499. Abscess.†
 (28.) *Pyæmic inflammation and ab-*
 500. Acute atrophy. [*sciss.*]
 501. Simple enlargement. *Syn.*,
 Congestion of the liver.
 502. Thickening of the capsule.
 503. Cirrhosis.
 504. Fatty liver.
 506. Fibroid deposit.
 505. Lardaceous liver. *Syn.*, Amy-
 loid disease of the liver.
 Waxy liver.‡
 (48.) *Syphilitic deposit.*
 (49.) *Cancer.*
 (50.) *Colloid.*
 507. Non-malignant tumor.
 508. Cyst.
 (531.) *Tubercle.*
 509. Hydatid.
 (1061-63.) *Injuries.*
 510. Jaundice. *Syn.*, Icterus.

511. Obstruction of vena portæ.
 DISEASES OF THE HEPATIC DUCTS
 AND GALL-BLADDER.

512. Inflammation.
 513. Ulcer.
 514. Perforation.
 a. Biliary fistula.
 515. Obstruction.
 (49.) *Cancer.*
 516. Gallstones.
 a. Passage of gallstones
 through duct.
 (1061-63.) *Injuries.*

DISEASES OF THE PANCREAS.

517. Abscess.
 518. Obstruction of the duct.
 (491.) *Cancer.*
 (501.) *Colloid.*
 519. Calculi.

DISEASES OF THE SPLEEN.

520. Splenitis.
 521. Abscess. [*sciss.*]
 (28.) *Pyæmic inflammation and ab-*
 522. Congestion. *Syn.*, Ague cake.
 523. Fibrinous deposit.
 524. Hypertrophy.
 a. Leucocythæmia.
 525. Lardaceous spleen. *Syn.*, Amy-
 loid disease. Waxy spleen.
 (491.) *Cancer.*
 (501.) *Colloid.*
 (531.) *Tubercle.*
 526. Hydatid.
 (1058.) *Rupture.*

DISEASES OF THE PERITONEUM.

527. Peritonitis.
 (713.) *a.* *Puerperal peritonitis.*
 b. *Chronic peritonitis.*
 c. *Suppurative peritonitis.*
 (531.) *d.* *Tubercular peritonitis.*
 e. *Adhesions of peritoneum.*
 528. * Ascites.
 (491.) *Cancer.*
 (501.) *Colloid.*
 529. Hydatid.
 (1059-62.) *Injuries.*

DISEASES OF THE MESENTERIC GLANDS.

530. Inflammation.
 531. Abscess.
 532. Enlargement.
 (491.) *Cancer.*
 533. Non-malignant growth.
 (531.) *Tubercle.*
 (531.) *Tabes mesenterica.*

DISEASES OF THE URINARY SYSTEM.

DISEASES OF THE KIDNEY.

534. Bright's disease. *Syn.*, Albu-
 minuria.
 1. Acute Bright's disease.
 Syn., Acute albuminuria.
 Acute desquamative
 nephritis. Acute renal
 dropsy.
 2. Chronic Bright's disease.
 Syn., Chronic albumin-
 uria.
 Sub-divisions:
 a. Granular kidney. *Syn.*,
 Contracted granular
 kidney. Chronic des-
 quamative nephritis.
 Gouty kidney.
 b. Fatty kidney.
 c. Lardaceous kidney.
 Syn., Amyloid disease.
 Waxy disease.

535. Suppurative nephritis.
 536. Abscess.
 537. Pyelitis.
 538. Fibrinous deposit.
 539. Hydronephrosis.
 540. Hypertrophy.
 541. Atrophy.
 (491.) *Cancer.*
 542. Non-malignant tumor.
 543. Simple cyst.
 544. Urinary cyst (from injury).
 (531.) *Tubercle.*
 545. Entozoa.
 a. Hydatid.
 b. *Eustrongylus gigas.*
 546. Calculus.
 547. Calculus in the ureter.
 548. Malformations.
 (1061-63.) *Injuries.*
 549. * Hæmaturia renalis.

550. * Suppression of urine. *Syn.*,
 Ischuria renalis.
 (47.) *Diabetes.* *Syn.*, Diabetes mel-
 551. * Diuresis. [litus.
 552. Movable kidney.

DISEASES OF THE BLADDER.

553. Cystitis. *Syn.*, Catarrh of
 the bladder.
 a. Acute.
 * *b.* Chronic.
 554. Ulceration.
 555. Suppuration.
 556. Sloughing.
 557. Vesico-intestinal fistula.
 558. Recto-vesical fistula.
 669. Vesico-vaginal fistula.
 559. Hypertrophy.
 560. * Distension.
 a. Sacculated bladder.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When abscess of the liver is associated with dysentery, injury, or any other condition, the fact should be stated.

‡ Such cases have been described under the name of Scrofulous disease of the liver.

- b. Rupture.
 561. Inversion.
 562. Extroversion.
 563. Hernia.
 (49.) *Cancer*.
 564. Fibrous tumor.
 565. Villous tumor.
 566. Calculus.
 a. Uric acid.
 b. Urate of ammonia.
 c. Uric oxide. *Syn.*, Xanthic oxide.
 d. Oxalate of lime.
 e. Cystic oxide.
 f. Phosphate of lime.
 g. Triple phosphate.
 h. Fusible.
 i. Carbonate of lime.
 k. Fibrinous.
 l. Urosteolith.
 m. Blood calculus.
 Foreign bodies.
 567. * Hæmaturia (vesical).
 568. * Paralysis.
 569. * Irritability.
 570. * Spasm.
 571. Neuralgia.
 572. * Incontinence of urine.

573. * Retention of urine.

DISEASES OF THE PROSTATE GLAND.

574. Inflammation.
 a. Acute.
 b. Chronic.
 575. Ulceration.
 576. Abscess.
 577. Atrophy.
 (49.) *Cancer*.
 578. Non-malignant tumor. *Syn.*, Enlarged lobe of prostate.
 579. Cyst.
 (71.) *Tubercle*.
 580. Calculi.

GONORRHOEA AND ITS COMPLICATIONS.

581. Gonorrhœa.
 a. In male.
 b. In female.
 582. Balanitis.
Herpes preputialis.
 583. Phimosis.
 584. Paraphimosis.
 585. Condyloma.
 a. In male.
 b. In female.

586. Gleet.
 587. Lacunar abscess.
 588. Bubo.
 Prostatic abscess.
 589. Epididymitis. *Syn.*, Gonorrhœal orchitis.
 a. Abscess.
 590. Abscess of the spermatic cord.
 (628.) *Inflammation of ovary*.
 591. Abscess of the vulva.
 (117.) *Gonorrhœal ophthalmia*.
 (140.) *Gonorrhœal iritis*.
 (36.) *Gonorrhœal rheumatism*.

DISEASES OF THE MALE URETHRA.

592. Stricture.†
 a. Organic.
 b. Traumatic.
 c. Spasmodic.
 d. Inflammatory.
 593. Ulcer.
 594. Urinary abscess.
 595. Urinary fistula.
 596. Recto-urethral fistula.
 597. Extravasation of urine.
 598. Impacted calculus.
 a. Foreign bodies.

DISEASES OF THE GENERATIVE SYSTEM.

DISEASES OF THE MALE ORGANS OF GENERATION.

PENIS.

599. Inflammation.
 600. Abscess.
 (581.) *Gonorrhœa*.
 601. Gangrene.
 602. * Priapism.
 (48.) *Syphilis*.
 (49.) *Cancer*.
 a. *Of prepuce*.
 b. *Of body*.
 603. Non-malignant tumor.
 (1069.) *Injuries*.
 604. Phimosis—congenital.

SCROTUM.

605. Sloughing.
 606. Edema.
 607. Elephantiasis.
 (828.) *Prurigo*.
 (48.) *Syphilis*.

- (49.) *Cancer*.
 (49.) *Epithelial cancer*. *Syn.*, *Chimney-sweeper's cancer*.
 608. Non-malignant tumor.

CORD.

609. Hydrocele.
Varieties:
 a. Encysted.
 b. Diffused.
 610. Varicocele.
 611. Non-malignant tumor.
 612. Neuralgia.

TUNICA VAGINALIS.

613. Inflammation.
 614. Hydrocele.
Varieties:
 a. Congenital.
 b. Infantile.
 c. Encysted.

615. Hæmatocele.
 616. Loose bodies.

TESTICLE.

617. Orchitis.
 a. Acute.
 b. Chronic.
 618. Abscess.
 619. Protrusion of tubuli. *Syn.*, *Hernia testis*. *Fungus testis*.
 620. Atrophy.
 (49.) *Cancer*.
 621. Non-malignant tumor.
 622. Cystic disease.
 (532.) *Tubercle*.
 (1069.) *Injuries*.
 623. Fœtal remains in testicle.
 624. Malposition.
 625. Spermatorrhœa.
 626. Impotence.
 627. Neuralgia.

DISEASES OF THE FEMALE ORGANS OF GENERATION IN THE UNIMPREGNATED STATE.

OVARY.

628. Inflammation.
 629. Abscess.
 630. Hæmorrhage.
 631. Atrophy.
 632. Hypertrophy.
 (49.) *Cancer*.
 633. Fibrous tumor.
 634. Encysted dropsy.
 635. Complex cystic tumors. *Syn.*, Alveolar, gelatinous, and colloid tumor of the ovary. Cysto-sarcoma.
 a. With intracystic growths.
 636. Cysts, containing tegumentary structures—hair, teeth, and bones. *Syn.*, Dermoid cysts.
 (53.) *Tubercle*.
 637. Cyst containing hydatid.
 638. Dislocation.
 639. Hernia.

FALLOPIAN TUBE.

640. Abscess.
 641. Dropsy.

642. Stricture.
 643. Occlusion.
 (49.) *Cancer*.
 644. Cyst.
 (53.) *Tubercle*.
 645. Dislocation.
 646. Hernia.

BROAD LIGAMENT.

647. Inflammation.
 a. Pelvic peritonitis.
 b. Pelvic cellulitis.
 648. Abscess.
 649. Cyst. [tocele.
 (817.) Peri-uterine or pelvic hæma-

UTERUS.

650. Catarrh. *Syn.*, Leucorrhœa.
 a. Hydrorrhœa.
 651. Inflammation.
 Granular inflammation.
 Abrasion.
 652. Ulcer.
 653. Rodent ulcer.
 654. Abscess.
 655. Utero-vesical fistula.

656. Stricture of the orifice.
 657. " of the canal.
 658. Occlusion of the orifice.
 659. " of the canal.
 660. Hypertrophy.
 661. Atrophy.
 (49.) *Cancer*.
 a. *Scirrhus*.
 b. *Medullary cancer*.
 c. *Epithelial cancer*.
 662. Fibrous tumor.
 663. Polypus.†
 (53.) *Tubercle*.
 664. Displacements and distortions.
 a. Anti-version.
 b. Retro-version.
 c. Anti-flexion.
 d. Retro-flexion.
 e. Inversion.
 f. Prolapsus.
 1. Procidentia.
 g. Hernia.

VAGINA.

665. Catarrh. *Syn.*, Leucorrhœa.
 666. Inflammation.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When the cause of the stricture is known it should be stated.

‡ Under this head should be returned all pedunculated tumors growing from the cavity or neck of the uterus, whether mucous, cellular, or fibrous.

667. Abscess.
(580^b.) *Gonorrhœa*.
668. Cicatrix or band.
668. Vaginal fistula.
669. Vesico-vaginal fistula.
670. Recto-vaginal fistula.
671. Hernia.
 a. Cystocele.
 b. Rectocele.
(491.) *Cancer*.
672. Laceration.
673. Polypus.

VULVA.

673. Inflammation of labia.
674. Pruritus of labia.
(853.) *Eczema of labia*.
675. Edema of labia.

676. Abscess.
677. Gangrene.
678. Hypertrophy.*
 a. Elongation of cervix.
679. Occlusion.
680. Imperforate hymen.
(262.) *Varicose veins*.
(481.) *Syphilis*.
(491.) *Cancer*.
681. Vascular tumor of the meatus
 urinaris.
682. Mucous cyst.
(585^b.) *Condyloma*.

FUNCTIONAL DISEASES.

683. Amenorrhœa. *English syn.*,
 Absent menstruation.

Varieties:

a. From original defective
 formation.
b. From want of develop-
 ment at the time of
 puberty.
c. From mechanical ob-
 struction.
d. From temporary sup-
 pression. [*sickness*.]
688. *Chlorosis*. *English syn.*, *Green*
684. Scanty menstruation. *Syn.*,
 Deficient menstruation.
685. Vicarious menstruation.
686. Dysmenorrhœa. *English syn.*,
 Painful menstruation.
687. Menorrhagia. *English syn.*,
 Excessive menstruation.

AFFECTIONS CONNECTED WITH PREGNANCY.

† DISORDERS OF THE NERVOUS SYSTEM.

Neuralgia.
Varieties:
 a. Odontalgia.
 b. Cephalalgia.
 c. Mastodynia.

Chorea.
Convulsions.
Hypochondriasis.
Mania.

† DISORDERS OF THE CIRCULATORY SYSTEM.

Varicose veins—
 a. Of the lower extremities.
 b. Of the labia.
 c. Of the rectum. Hæmor-
Seros exudation. [rhoids].
Varieties:
 a. Ascites.
 b. Edema of labia.
 c. Edema of lower ex-
Syncope. [tremities].
Palpitation.

† DISORDERS OF THE RESPIRATORY SYSTEM.

Dyspnoea.
Orthopnoea.
Cough.

† DISORDERS OF THE DIGESTIVE SYSTEM.

Salivation. [petite].
Depraved and capricious ap-
Nausea and vomiting.
Cardialgia or Heartburn.
Pyrosis.
Intestinal cramp—colic.
Constipation.
Diarrhœa.
Jaundice.

† DISORDERS OF THE URINARY SYSTEM.

Albuminuria.
Dysuria.
Incontinence of urine.
Retention of urine.

DISORDERS OF THE GENERATIVE SYSTEM.

689. Metritis. *Syn.*, Hysteritis.
690. Discharge of watery fluid from
 the uterus. Hydrorrhœa.
691. Rheumatism of the uterus.
692. Hysteralgia.
693. Spurious pain and cramps.
(665.) *Catarrh of vagina*. *Syn.*,
 Leucorrhœa.
694. Sanguineous discharge. *Syn.*,
 Menstruation.
695. Hæmorrhage.
696. Displacements of uterus.

Varieties:

a. Prolapsus.
b. Hernia.
c. Retro-version.
(674.) *Pruritus of the vulva*.
697. Abortion.
698. Premature labour.
699. Extra-uterine gestation.

AFFECTIONS CONNECTED WITH PARTURITION.

700. Atony of the uterus.
701. Over-distension of the uterus.
 a. From excess of liquor
 amni.
 b. " twins, triplets, &c.
702. Mechanical obstacle to the
 action of the uterus. [uteri].
 a. From occlusion of the os
 b. From rigidity of the—
 os uteri.
 vagina.
 perineum.
 c. From cancer of the cervix
 uteri.
 d. From narrowness of the
 vagina.
 e. From cicatrix or band in
 the vagina.
 f. From vaginal cyst.
 g. From prolapsus of the
 bladder.
 h. From stone in the bladder.
 i. " distended rectum.
 k. " prolapsus of the rec-
 l. " tumor. [tum].

Varieties:

1. Uterine.
2. Ovarian.
3. Pelvic.
4. Of external parts.
m. From polypus.
n. " fractured pelvis.
o. " exostosis.
p. From distorted or con-
 tracted pelvis.
q. From dislocated lumbar
 vertebræ into pelvis.
 Syn., Spondylolisthesis.
r. From ankylosed coccyx.
s. From diminutive pelvis.
t. Extreme ante-version of
 uterus (with pendulous
 abdomen).
u. From excessive size of
 fœtus.
v. From malposition of fœtus.
w. From malformation of
 fœtus.
x. From enlargement of
 fœtus from disease.
y. From unusual thickness
 of fœtal membranes.
z. From unusual shortness
 of funis.

703. Hæmorrhage.

a. From placenta prævia.
 Syn., Unavoidable hæ-
 morrhage.
b. From accidental detach-
 ment of placenta. *Syn.*,
 Accidental hæmorrhage.
c. Thrombus of cervix uteri
 or labium.

704. Rupture or laceration of the—
uterus.

705. vagina.
706. urinary bladder.
707. perineum.

708. Retention of the placenta.

a. From atony of the uterus.
b. From irregular or hour-
 glass contraction.
c. From preternatural adhe-
 sions.

709. Inversion of the uterus.

710. Convulsions.

AFFECTIONS CONSEQUENT ON PAR-
TURITION.

711. Post-partum hæmorrhage.

(33.) *Puerperal ephemera*.

712. Milk fever.

(32.) *Puerperal fever*.

713. Metro-peritonitis. *Syn.*, Puer-
peral peritonitis.

a. Metritis.
b. Peritonitis.

(257.) *Phlebitis*.

(258.) *Phlegmasia dolens*.

714. Pelvic cellulitis.

715. Iliac and pelvic abscesses.

716. Sloughing of cervix uteri.

717. " vagina.

718. " perineum.

719. " bladder.

720. " rectum.

(655.) *Utero-vesical fistula*.

(669.) *Vesico-vaginal fistula*.

(670.) *Recto-vaginal fistula*.

(723.) *Mammary inflammation*.

(724.) *Mammary abscess*.

721. Puerperal mania. [tion].

a. Connected with parturi-
 lactation.
b. " "

722. Puerperal convulsions. *Syn.*,
Eclampsia.

723. Sudden death after delivery.

a. From shock or nervous
 exhaustion.

b. From impaction of coa-
 gula in the heart and
 great vessels.

1. Thrombosis.
2. Embolism.

c. From entrance of air into
 veins (from separa-
 tion of placenta).

(896.) *Still-born*.

(897.) *Premature birth*.

* Specify the part.

† These affections are secondary, and are therefore not numbered.

DISEASES OF THE FEMALE BREAST.

- | | | |
|--|--|---|
| 723. Inflammation.
<i>a.</i> Acute.
<i>b.</i> Chronic.
724. Abscess.
725. Sinus.
726. Galactorrhœa.
727. Deficiency of milk.
728. Hypertrophy.
729. Atrophy.
730. Depressed nipple.
731. Chapped nipple. | 732. Ulcerated nipple.
(49.) <i>Cancer.</i>
<i>a.</i> <i>Scirrhus.</i>
<i>b.</i> <i>Medullary cancer.</i>
<i>c.</i> <i>Epithelial cancer.</i>
(50.) <i>Colloid.</i>
733. Fibrous tumor. <i>Syn., Painful</i>
subcutaneous tumor.
734. Fibro-plastic tumor.
735. Fatty tumor.
736. Osseous tumor. | 737. Cartilaginous tumor. <i>Syn.,</i>
Enchondroma.
738. Chronic mammary tumor.
<i>Syn., Adenoid tumor.</i>
739. Vascular tumor.
740. Cyst.
741. Complex cystic tumor. <i>Syn.,</i>
Cysto-sarcoma.
742. Hydatid.
743. Hyperæsthesia.
744. Mastodynia. <i>Syn., Neuralgia.</i> |
|--|--|---|

DISEASES OF THE MALE MAMMILLA.

- | | | |
|---|---|------------|
| 745. Inflammation.
746. Hypertrophy. | (49.) <i>Cancer.</i>
747. Non-malignant tumor. | 748. Cyst. |
|---|---|------------|

DISEASES OF THE ORGANS OF LOCOMOTION.

DISEASES OF BONES.*

- | | | |
|--|---|--|
| 749. Ostitis.
<i>a.</i> Periostitis.
1. Nodes.
750. Diffuse periostitis. <i>Syn., Acute</i>
periosteal abscess.
<i>a.</i> Acute necrosis.
751. Osteo-myelitis.
752. Chronic abscess.
753. Caries.
754. Necrosis.
755. Mollities ossium.
756. Hypertrophy.
757. Atrophy.
758. Spontaneous fracture. (The
cause, if known, should be
stated.)
(48.) <i>Syphilitic disease.</i>
(49.) <i>Cancer.</i>
759. Non-malignant tumors.
<i>a.</i> Fibrous and fibro-cystic.
<i>b.</i> Myeloid.
<i>c.</i> Cartilaginous. <i>Syn., En-</i>
chondroma.
<i>d.</i> Exostosis.
1. Diffused exostosis.
760. Cyst.
(54.) <i>Rickets.</i>
(53.) <i>Scrofulous disease.</i>
761. Hydatid. | (43.) <i>Chronic osteo-arthritis.</i> <i>Syn.,</i>
<i>Chronic rheumatic arthritis.</i>
768. Degeneration of cartilage and
articular surfaces of bones.
769. Perforation of joints.†
770. Loose cartilage. <i>Syn., Loose</i>
body.
771. Relaxation of ligaments.
772. Displacement of articular car-
773. Knock-knee. [tilage.
774. Bow-leg, or out-knee.
(49.) <i>Cancer.</i>
775. Non-malignant tumor.
776. Neuralgia of joints. | 773. Ossification.
(48.) <i>Syphilitic deposit.</i>
(49.) <i>Cancer.</i>
(50.) <i>Colloid.</i>
794. Non-malignant tumor.
<i>a.</i> Erectile tumor.
795. Cyst.
<i>Rupture.</i>
(86.) <i>Infantile paralysis.</i>
796. Trichinosis.
(93.) <i>Spasm.</i>
797. Exhaustion.
(87.) <i>Scrivener's palsy.</i>
(19.) <i>Diphtheritic paralysis.</i> |
|--|---|--|

DISEASES OF THE SPINE.

TENDONS.

- | | | |
|---|--|---|
| DISEASES OF JOINTS.†
762. Acute synovitis.
763. Chronic synovitis.
<i>a.</i> Pulpy degeneration of
synovial membrane.
<i>b.</i> <i>Strumous disease of joints.</i>
1. <i>Morbus coræ.</i>
764. Ulceration of cartilage.
765. Abscess.
<i>a.</i> <i>Pyæmic abscess.</i>
766. Ankylosis.
<i>a.</i> Deformity from ankylosis.
767. Dropsy of joint.
(36.) <i>Gonorrhœal rheumatism.</i>
(37.) <i>Synovial rheumatism.</i>
(42.) <i>Gouty synovitis.</i> | 777. Ulceration of ligaments and
cartilages.
778. Caries and necrosis.
<i>a.</i> Spontaneous fracture of
odontoid process.
779. Psoas, lumbar, and other ab-
scesses.
780. Angular deformity. <i>Syn., Ky-</i>
phosis.
781. Lateral curvature. <i>Syn., Sko-</i>
liosis.
782. Anterior curvature. <i>Syn.,</i>
Lordosis.
(54.) <i>Rickety curvature.</i>
783. Ankylosis.
(43.) <i>Chronic osteo-arthritis.</i>
784. Non-malignant tumor.
(49.) <i>Cancer.</i>
785. Hydatid. [tions.
786. Deformity from malforma- | 798. Inflammation.
(875.) <i>Thecal abscess.</i>
799. Adhesion of tendons.
(49.) <i>Cancer.</i>
800. Non-malignant tumors.
801. Contraction of tendons, fasciæ,
or muscles.
802. Club-foot.
<i>a.</i> Talipes varus.
<i>b.</i> " valgus.
<i>c.</i> " equinus.
<i>d.</i> " calcaneus.
<i>e.</i> " calcaneo-varus.
<i>f.</i> " equino-valgus.
<i>Syn., Flat-foot.</i>
803. Club-hand.
804. Contracted palmar fascia.
805. Wry neck.
(1135.) <i>Rupture of tendon.</i> |
|---|--|---|

DISEASES OF THE MUSCULAR
SYSTEM.§

MUSCLES.

APPENDAGES OF MUSCULAR SYSTEM.

DISEASES OF THE CELLULAR TISSUE.

- | | | |
|---|---|--|
| 812. Inflammation.
813. Abscess.
814. Inflammatory induration in
the newly born.
815. Slough. | (27b.) <i>Phlegmonous erysipelas.</i>
(86.) <i>Carbuncle.</i> <i>Syn., Anthrax.</i>
816. Obesity.
817. Hæmorrhage.
<i>a.</i> Pelvic hæmatocele. | 818. Non-malignant tumor.
(49.) <i>Cancer.</i>
819. Parasites.
(135.) <i>Foreign substances.</i>
820. Emphysema. |
|---|---|--|

DISEASES OF THE CUTANEOUS SYSTEM.

- | | | |
|---|---|--|
| DISEASES OF THE SKIN.
(27.) <i>Erysipelas.</i> | 821. Erythema. (This term includes
1. Erythema læve. | 2. Erythema fugax. <i>Syn., E.</i>
volaticum. |
|---|---|--|

* In all cases the bone affected must be specified.

† In all cases the joint affected is to be specified.

‡ This refers to perforation by disease, and should be returned with the original affection.

§ In all cases the affected muscle or muscles should be stated.

|| Where the disease is local, its situation should be specified.

3. Erythema marginatum.
4. " papulatum.
5. " tuberculatum.
6. " nodosum.)
822. Intertrigo.
823. Roseola. (This term includes
1. Roseola æstiva.
2. " autumnalis.
3. " symptomatica.
4. " annulata.)
824. Urticaria. *English Syn.*, Nettle
a. Urticaria acuta. [rash.
b. " chronica.
- (Under one or other of these heads
are included
1. Urticaria febrilis.
2. " evanida.
3. " perstans.
4. " conferta.
5. " subcutanea.
6. " tuberculata.)
825. Pellagra.
826. Acrodynia.
827. Asturian rose.
828. Prurigo.
829. Lichen. (This term includes
1. Lichen simplex.
2. " pilaris.
3. " circumscriptus.
4. " agrius.
5. " tropicus, *English
syn.*, Prickly heat.)
(The so-called Lichen lividus
is really a form of Purpura.)
830. Strophulus. *English syn.*, Red
gum. Tooth rash. (This term
includes
1. Strophulus intertinctus.
2. " confertus.
3. " candidus.)
(Strophulus albidus is referred
to Acne.
" volaticus to Erythema.)
831. Pityriasis. (This term includes
Pityriasis capitis. *English
syn.*, Dandriff.)
(Pityriasis versicolor is re-
ferred to Parasitic affections
as a *Synonym* of Tinea ver-
sicolo.)
832. Psoriasis. (This term includes
Lepra.)
833. Psoriasis vulgaris. *Syn.*, Lepra
vulgaris.
a. Psoriasis guttata.
b. " diffusa.
c. " gyrata.
d. " inveterata.
834. Miliaria.
a. Sudamina.
835. Herpes.
a. Herpes phlyctænodes.
b. " circinatus.
c. " iris.
d. " zoster. *English
Syn.*, Shingles.
836. Pemphigus. *Syn.*, Pompholyx.
a. Pemphigus acutus.
b. " chronicus.
c. " solitarius.
837. Eczema.
a. Eczema simplex.
b. " rubrum.
c. " impetiginodes.
d. " chronicum.
838. Impetigo.
a. Impetigo sparsa.
b. " confluens.
1. figurata.
2. larvalis.
839. Rupia.
a. Rupia simplex.
b. " prominens.
c. " escharotica. *Syn.*,
Pemphigus gan-
grænosus.
840. Ecthyma.
841. Achne.
a. Achne punctata.*
b. " indurata.
c. " rosacea.
d. " strophulosa. *Syn.*,
Strophulus al-
bidus.
842. Sycosis. *Syn.*, Mentagra.†
843. Stearrhœa.
a. Stearrhœa simplex.
b. " nigricans.
844. Ichthyosis.
a. Ichthyosis vera.
b. " cornea.
845. Xeroderma.
846. Leucoderma. (This term in-
cludes Vitiligo.)
847. Albinismus.
848. Canities.
849. Melasma.
(282.) *Melasma Addisoni. English
Syn.*, Addison's disease.
Bronze skin.
850. Lentigo and Ephelis. *English
Syn.*, Freckles.
851. Chilblain.
852. Frostbite.
853. Ulcer.
854. Fissures.
(352.) *Cancerum oris.*
855. Boil.
856. Carbuncle. *Syn.*, Anthrax.
(26.) *Malignant pustule.*
857. Onychia.
858. Onychia maligna.
859. Whitlow.
a. Thecal abscess.
860. Gangrene.
861. Hypertrophy.
862. Corn.
863. Bunion.
864. Elephantiasis arabum. *Syn.*,
Barbadoes leg; Elephas.
(51.) True leprosy. *Syn.*, El. Græ-
corum.
865. Atrophy.
a. Linear atrophy.
b. Alopecia.
c. Atrophy of nails.
(49.) *Cancer.*
866. Fibro-cellular tumor.
867. Fatty tumor.
(263.) *Nævus vascularis.*
868. Nævus. *Syn.*, Port wine stain.
869. Nævus pilaris. *Syn.*, Mole.
870. Sebaceous tumor.
a. Steatoma.
871. Cornua.
872. Molluscum.
873. Warts.
874. Condyloma.
a. Male.
b. Female.
875. Cheloid.
876. Framboesia.
(51.) *Lupus.*
(53.) *Scrofulous disease.*
877. Ingrown nail.
878. Silver-stain.
(986.) *Burns and scalds.*
(101.) *Hyperæsthesia.*
879. Pruritus.
(102.) *Anæsthesia.*
880. Ephidrosis.
881. Anidrosis.
- PARASITIC DISEASES OF THE SKIN.
882. Tinea tonsurans. *Syn.*, Ring-
worm. *Parasite, Trichophy-
ton tonsurans.*
883. Tinea decalvans. *Syn.*, Alo-
pecia areata. *Par.*, Micro-
sporon Audouini.
884. Tinea favosa. *Syn.*, Favus.
Par., Achorion Schönleini,
Pucciani Favi.
885. Tinea versicolor. *Syn.*, Pity-
riasis versicolor. *Par.*,
Microsporon furfur.
886. Tinea Polonica. *Syn.*, Plica
Polonica. *Par.*, Trichophy-
ton sporuloides.
887. Mycetoma. *Syn.*, Madura foot.
Par., Chionyphe Carteri.
888. Scabies. *Syn.*, Itch. *Par.*, Sar-
coptes scabiei.
889. Phthiriasis from Phthirius
inguinalis. *English Syn.*,
Crab-louse.
890. Irritation caused by
a. Pediculus capitis.
b. " palpebrarum.
c. " vestimentum.
d. " tabescentium.
891. Pulex penetrans. *English
Syn.*, Chigoe.
" irritans.
892. Cimex.
893. Leptothrix autumnalis. *Eng-
lish Syn.*, Harvest-bug.
894. Wasps, bees, and other sting-
ing insects.
895. Nettles and other stinging
plants
- CONDITIONS NOT NECESSARILY AS-
SOCIATED WITH GENERAL OR
LOCAL DISEASE.
896. Still-born.
897. Premature birth.
898. Old age.‡
899. § Debility.

POISONS.

In returning cases of poisoning, the precise agent should be stated.

- METALS AND THEIR SALTS.
900. Arsenic.
901. Mercury.
(96.) *Mercurial tremor.*
902. Lead.

- a. Lead colic. *Syn.*, Painters'
colic.
b. Lead palsy.
c. Blue gum.
d. Metallic stain of conjunc-
tiva (from lead).
903. Copper.
904. Antimony.
905. Zinc.
906. Silver.
a. Silver-stain.
907. Iron.

* When the Demodex folliculorum is discovered, its presence should be stated.

† When the Microsporon mentagrophytes is discovered, its presence should be stated.

‡ This mode of return is only to be employed when the cause of death is not traceable to definite disease.

§ When the cause of this affection has been ascertained, the case should be returned under the head
of the primary disease, the secondary affection being also specified.

908. Bismuth.
909. Chromium.
 a. (Bichromate of potash.)

CAUSTIC ALKALIES.

910. Potash.
911. Soda.
912. Ammonia.
913. Alkaline salts.

METALLOIDS.

914. Phosphorus.
915. Iodine.
 ACIDS.
916. Sulphuric acid.
917. Nitric acid.
918. Muriatic acid.
919. Phosphorous acid.
920. Oxalic acid.
921. Tartaric acid.

VEGETABLE POISONS.

922. Savin.
923. Croton oil.
924. Elaterium.
925. Colchicum.
926. Black hellebore.
927. White hellebore.
 a. Veratria.
928. Squill.
929. Ergot of rye.
(46.) *a.* Ergotism.
930. Opium.
931. Indian hemp. (*Cannabis indica*.)
932. Alcohol. [dica.]
(103.) *a.* Delirium tremens.
933. Ether vapour.
934. Chloroform vapour.
935. Henbane, (*Hyoscyamus*.)
936. Deadly nightshade. (*Belladonna*.)
 a. Atropia.

937. Thorn apple. (*Stramonium*.)
938. Prussic acid.
 a. Oil of bitter almonds.
 b. Laurel water.
939. Cyanide of potassium.
940. Nitro-benzole.
941. Wourali. Curara. (*Strychnos toxifera*.)
942. Hemlock. Conium.
943. Monkshood. (*Aconitum*.)
 a. Aconitia.
944. Foxglove. (*Digitalis*.)
 a. Digitalin.
945. Tobacco.
 a. Nicotine.
950. Hemlock dropwort. (*Eranthis crocata*.)
946. Nux vomica. [crocata.]
 a. Strychnia.
 b. Brucia.
947. Upas tieute. (*Strychnos tieute*.)
948. Upas antiar. [tieute.]
949. Calabar bean. (*Physostigma venenosum*.)
951. Fool's parsley. (*Æthusa cynapium*.)
952. Water hemlock. (*Cicuta virosa*.)
953. Camphor. [virosa.]
954. Cocculus Indicus. [tum.]
955. Darnel. (*Lolium temulen-*
956. Indian tobacco. (*Lobelia inflata*.)
957. Laburnum. [flata.]
958. Yew. (*Taxus baccata*.)
959. Poisonous fungi.
 a. Mouldy bread.

ANIMAL POISONS.

960. Spanish fly. (*Cantharides*.)
961. Decayed and diseased meat.
962. Poisonous meat.
 a. Sausages.
963. Poisonous cheese.
964. Poisonous milk.

965. Poisonous fish.
 a. Mussels.

GASEOUS POISONS.

966. Ammonia.
967. Nitrous acid vapour.
968. Chlorine.
969. Carbonic acid.
970. Carbonic oxide.
971. Coal gas.
972. Cyanogen.
973. Sulphuretted hydrogen.
974. Putrid and morbid exhalations.
975. Other noxious effluvia.

MECHANICAL IRRITANTS.

976. Pounded glass.
977. Steel filings.

POISONED WOUNDS.

Varieties:

979. *a.* By venomous animals.
 1. Snakes.
 2. Scorpions.
 3. *Stinging insects*.
(894.) *b.* By animals having infectious disease.
(23.) Glanders.
(24.) Farcy.
(25.) *Equinia mitis*.
(26.) Malignant pustule.
(89.) Hydrophobia.
(2.) Cowpox.
980. *c.* By dead animal matter.
981. *d.* By morbid secretions.
982. *e.* By vegetable substances.
983. 1. Poisoned arrows.
 Wourali.
984. 2. Subcutaneous injection.*
985. *f.* By mineral substances.

INJURIES.

GENERAL INJURIES.

986. Burns and scalds (including explosions).†
987. Lightning stroke.
988. Multiple injury. (The cause and extent to be stated.)

989. Asphyxia.

- a.* Drowning.
b. Hanging.
c. Strangling.
d. Overlying.

e. Gaseous Poisons.

990. Privation.‡
991. Exposure to cold.‡
992. Infant exposure.‡
993. Neglect.‡

LOCAL INJURIES.§

INJURIES OF THE HEAD AND FACE.

A.—OF THE HEAD.

994. Contusion.
 a. Cephalhæmatoma.
995. Scalp-wound: bone not exposed.
996. " bone exposed.
997. Concussion of the brain.
998. Fracture of the vault of the skull.
 a. Simple, without depression.
 b. " with depression.
 c. Compound, without depression.¶
 d. " with depression.
999. Hernia cerebri.
1000. Fracture of the base of the skull.
1001. Wound of the skull.¶
1002. Laceration of the brain, without fracture.

1003. Injuries of the vessels. (Specify which.)
1004. Injuries of the cerebral nerves.

B.—OF THE FACE.

1005. Contusion.
1006. Wound.¶
1007. Foreign bodies in the ear.
1008. " nose.
1009. " antrum.
1010. " soft parts.
1011. Fracture of the facial bones.
1012. " lower jaw.**
1013. Dislocation of the jaw.

INJURIES OF THE EYE.

1014. Contusions.
1015. Contusion, with rupture of the sclerotic. *Syn.*, Ruptured globe.
1016. Contusion, with dislocation of the lens.

1017. Contusion, with hæmorrhage into the globe.
1018. Foreign bodies in the cornea or conjunctiva.
1019. Foreign bodies in the cavity of the eye.¶
1020. Wounds of the eyelids.
1021. " conjunctiva.
1022. " sclerotic.
1023. " cornea.
1024. " lens.
1025. " iris.
1026. Dislocation of the globe.
(153.) Total disorganization of the eye from injury.
1027. Wounds and injuries of the parts within the orbit.
1028. Chemical injuries of the eyelids and eye.
1029. Burns and scalds.
INJURIES OF THE NECK.
1030. Contusion of the soft parts.

* In returning such cases, specify the agent employed.

† When limited to one part of the body, the part is to be specified; *e. g.*, Scald of the larynx.

‡ Any affection that may have been induced by this cause ought to be stated.

§ In all cases of injury, specify whether accidental, judicial, homicidal, self-inflicted, or in battle.

¶ In such cases, state the main features of the case in the fewest words possible.

¶ If from gun-shot, to be so stated.

** Injuries of the alveoli and teeth are to be returned with the other affections of those parts.

1031. Fracture of the hyoid bone.	1062. Wound of parietes, with wound of unprotruded viscera.	1098. Fracture. (State whether simple or compound.)
1032. " cartilages of the larynx.	1063. Wound of viscera without wound of parietes.*	1099. " of clavicle.
1033. Rupture of the trachea.	1064. Foreign bodies in the peritoneal cavity.	1100. " scapula.
1034. Dislocation of the hyoid bone.	1065. Foreign bodies in the stomach.	1101. " humerus.
1035. Wound.	1066. Foreign bodies and concretions in the intestine.	1102. " forearm.
a. Superficial.	1067. Fistula from injury, and artificial anus.	1103. " carpus, metacarpus, and phalanges.
b. Cut throat.*		1104. Ununited fracture, or false joint. (Specify which bone.)
c. Gun-shot.*		
d. Of great vessels.*		
e. From the mouth.		
(986.) <i>Burn and scald of larynx.</i>		
1036. Foreign bodies in the air passages.	INJURIES OF THE PELVIS.	INJURIES OF THE LOWER EXTREMITIES.
1037. " " pharynx.	1068. Contusion.	1105. Contusion.
1038. " " œsophagus.	1069. Wound of the male perineum, scrotum, and penis.†	1106. Sprain. (Specify which joint.)
1039. Injury of pharynx and œsophagus by corrosive substances.	1070. Wound of the female perineum and vulva.	1107. Wound.*†
	1071. Wound of the vagina and internal female organs.*	1108. " of joint.
INJURIES OF THE CHEST.*	1072. Wound of the rectum.*	1109. " of vessels.*
1040. Contusion.	1073. " anus.	1110. Foreign bodies embedded.*
1041. Fracture of the ribs (including costal cartilages) without injury to lung.	1074. " bladder.	1111. Separation of epiphyses.
1042. Fracture of the ribs (including costal cartilages) with injury to lung.	1075. Rupture of bladder without wound.	Fracture. (When compound, to be so stated.)
1043. Fracture of sternum.	1076. Rupture of bladder from fracture.§ [uterus.	1112. " of femur.
1044. Wound of parietes.	1077. Injuries of the pregnant	1113. " of cervix femoris.
1045. Perforating wound of chest.*†	1078. Foreign bodies in vagina.	1114. " intra capsular.
1046. Perforating wound of pleura or lung.*†	1079. " " rectum.	1115. Fracture of trochanter major.
1047. Wound of anterior mediastinum.*†	(566, 598.) " " bladder and urethra.	1116. " patella.
1048. Wound of pericardium and heart.*†	1080. Fracture and dislocation of pelvis.	1117. " leg, both bones.
1049. Wound of vessels.*†	1081. Fracture and dislocation of pelvis, with rupture of bladder or urethra.	1118. " " tibia alone.
1050. Rupture of heart or lung without wound or fracture.*	INJURIES OF THE UPPER EXTREMITIES.	1119. " " fibula alone.
INJURIES OF THE BACK. (Including the whole spinal region.)	1082. Contusion.	1120. " bones of the foot.
1051. Contusion.	1083. Wound.*†	Dislocation. (When compound, to be so stated.)
1052. Sprain.	1084. Wound of joint.	1121. " of hip.
1053. Wound.*†	1085. " vessels.*	1122. " patella.
1054. Fracture and dislocation of spine.‡	1086. Sprain. (Specify which joint.)	1123. " knee.
1055. Injury of the chord without known fracture.	1087. Dislocation. (When compound, to be so stated.)	1124. " head of fibula.
INJURIES OF THE ABDOMEN.	1088. Dislocation of sterno-clavicular joint.	1125. " foot, at the ankle.
1056. Contusion.	1089. Dislocation of acromio-clavicular joint.	1126. " " at calcaneo-astragaloid, and scapho-astragaloid joints.
1057. Contusion with rupture of muscles.*	1090. Dislocation of shoulder.	1127. " astragalus.
1058. Contusion with rupture of viscera.	1091. Dislocation of elbow.	1128. " os calcis.
1059. Wound of parietes.†	1092. " wrist and carpus.	1129. " other tarsal bones.
1060. Wound of parietes, with protrusion of uninjured viscera.	1093. " thumb.	1130. " metatarsus, and phalanges.
1061. Wound of parietes, with protrusion of wounded viscera.	1094. " phalangeal joints.	1131. Ununited fracture, or false joint. (Specify which bone.)
	1095. Separation of epiphyses.	INJURIES OF THE ABSORBENT SYSTEM.
	1096. Foreign bodies embedded.*	1132. Foreign bodies and concretions.
	1097. Greenstick fracture, or bending of bone. (Specify which bone.)	1133. Wounds of lymphatics.
		1134. Rupture of muscle.
		1135. " tendon.
		1136. Foreign substances in cellular tissue.

HUMAN PARASITES.

The Parasites are to be returned under Local Diseases.

SUBDIVISIONS.

1. ENTOZOA.	CLASS A.—CŒLELMINTHA.	Filaria oculi. (Nordmann.) <i>Syn.</i> ,
2. ECTOZOA.	Ascaris lumbricoides. (Linnaeus.)	Filaria lentis. (Diesing.) <i>Hab.</i> ,
3. ENTOPHYTA AND EPIPHYTA.	<i>Habitat</i> , Intestines.	Eye.
	Ascaris mystax. (Rudolphi.) <i>Hab.</i> ,	Strongylus bronchialis. (Cobbold.)
	Intestines.	<i>Hab.</i> , Bronchial tubes.
ENTOZOA.	Trichocephalus dispar. (Rudolphi.) <i>Hab.</i> , Intestines.	Eustrongylus gigas. (Diesing.)
CLASSES.	Trichina spiralis. (Owen.) <i>Hab.</i> ,	<i>Hab.</i> , Kidney; intestines.
A. CŒLELMINTHA. <i>English Syn.</i> ,	Muscles.	Sclerostoma duodenale. (Cobbold.) <i>Syn.</i> , Anchylostomum
Hollow worms.	Filaria Medinensis. (Gmelin.)	duodenale. <i>Hab.</i> , Duodenum.
B. STERELMINTHA. <i>English Syn.</i> ,	<i>English syn.</i> , Guinea-worm. <i>Hab.</i> ,	Dactylius aculeatus. (Curling.)
Solid worms.	Skin and subcutaneous tissues.	<i>Hab.</i> , Bladder.
C. ACCIDENTAL PARASITES.		

* In such cases, state the main features of the case in the fewest words possible.

† Specify when from gun-shot.

‡ The seat of the injury, and the existence and extent of paralysis, to be stated.

§ Rupture of bladder from accumulation of urine is usually from stricture, and must be returned under the appropriate heading.

|| Return such cases with calculus in the bladder and urethra.

Oxyuris vermicularis. (Bremser.)
English syn., Thread-worm.
Hab., Rectum.

CLASS B.—STERELMINTHA.

Bothriocephalus latus. (Brémser.)
Hab., Intestines.

Bothriocephalus cordatus.

(Leuckart.) *Hab.,* Intestines.

Tænia solium. (Linnæus.) *Hab.,*

Intestines.

Cysticercus of Tænia solium, Syn.,

Cysticercus telæ cellulosæ.

Tænia mediocanellata. (Küchen-

meister.) *Hab.,* Intestines.

Tænia acanthotrias. (Weinland.)

Hab., Intestines.

Tænia flavopuncta. (Weinland.)

Hab., Intestines.

Tænia nana. (Siebold.) *Hab.,*

Intestines.

Tænia lophosoma. (Cobbold.)

Hab., Intestines.

Tænia elliptica. (Batsch.) *Hab.,*

Intestines.

Cysticercus of the Tænia margin-

ata. Syn., Cysticercus tenuicollis.

Echinococcus hominis, or Hydatid

of the Tænia echinococcus.

(Siebold.)

Fasciola hepatica. (Linnæus.)

Hab., Liver.

Distoma crassum. (Busk.) *Hab.,*

Duodenum.

Distoma lanceolatum. (Mehlis.)

Hab., Hepatic duct; bowels.

Distoma ophthalmobium. (Dies-

ing.) *Hab.,* Eye.

Distoma heterophyes. (Siebold.)

Hab., Small intestines.

Bilharzia hæmatobia. (Cobbold.)

Hab., Portal and venous blood.

Tetrastoma renale. (Della Chiaje.)

Hab., Tubes of kidney.

Hexathyridium venarum. (Treut-

ler.) *Hab.,* Venous blood.

Hexathyridium pinguicola.

(Treutler.) *Hab.,* Ovary.

CLASS C.—ACCIDENTAL PARASITES.

Pentastoma denticulatum. (Sie-

bold.) *Hab.,* Liver; small in-

testines.

Pentastoma constrictum. *Hab.,*

Liver.

Cæstrus hominis. (Say.) *English*

syn., Larva of the gad-fly,

Hab., Intestines.

Anthomyia canicularis. (A. Farre.)

Hab., Intestines.

ECTOZOA.

Phthirus inguinalis. (Leach.)

English syn., Crab-louse.

Pediculus capitis. (Nitzsch.)

Pediculus palpebrarum. (Le

Jeune in Guillemeau.)

Pediculus vestimenti. (Nitzsch.)

English syn., Body-louse.

Pediculus tabescentium. (Bur-

meister.)

Sarcoptes scabiei. (Latreille.) *Syn.,*

Acarus. English syn., Itch-insect.*

Demodex folliculorum. (Owen.)

Pulex penetrans. (Gmelin.) *Syn.,*

Chigoe.

ENTOPHYTA AND EPIPHYTA.

Leptothrix buccalis. (Wedl. Robin.) *English syn.,* Alga of mouth.

Oidium albicans. (Link.) *English syn.,* Thrush fungus. *Hab.,* Mouth in cases of thrush, and certain mucous and cutaneous surfaces.

Sarcina ventriculi. (Goodsir) *Hab.,* Stomach.

Torula cerevisiæ. (Turpin.) *Syn.,* *Cryptococcus cerevisiæ.* (Kütz- ing.) *English syn.,* Yeast plant. *Hab.,* Stomach, bladder, &c.

Chionyphe Carteri. *Hab.,* Deep tissues, and bones of the hands and feet.

Achorion Schönleini. (Remak.)

Hab., Tinea favosa.*

Puccinia favi. (Ardsten.) *Hab.,*

Tinea favosa.*

Achorion Lebertii. (Robin.) *Syn.,*

Trichophyton tonsurans. (Malm-

sten.) *Hab.,* Tinea tonsurans.*

Microsporon Audouini. (Gruby.)

Hab., Tinea decalvans.*

Trichophyton sporuloides. (Von

Walther.) *Hab.,* Tinea polonica.*

Microsporon furfur. (Eichstädt.)

Hab., Tinea versicolor.*

Microsporon mentagrophytes.

(Gruby.) *Hab.,* Follicles of hair

in Sycosis or Mentagra.*

The foregoing list might be extended by the addition of various parasitic vegetations, which have been reported under the names of Algæ, Fungi, Mycodermis, Leptomiti, &c., but the characters or the existence of which are still the subject of inquiry.

CONGENITAL MALFORMATIONS OF THE FŒTUS.

MALFORMATIONS RESULTING FROM INCOMPLETE DEVELOPMENT OR GROWTH OF PARTS.

OF THE BODY GENERALLY.

Head absent, or rudimentary.
Cranium defective.
Lower jaw absent or defective.
Upper and lower extremities absent.
Lower extremities absent.
One lower extremity absent.
Hands and feet articulated to scapulæ and pelvis.
Fingers and toes deficient in number.

OF THE NERVOUS SYSTEM.

Brain absent.
Brain rudimentary or incompletely developed.
Spinal cord absent or imperfect.
Continuity of nerves with nerve-centres incomplete.

OF THE ORGANS OF SPECIAL SENSE.

Eyes absent.
Eyes imperfect.
Eyelids incomplete. Eyelids remaining united, (Symblepharon.)
External ear absent. Pinna adherent.
Meatus externus closed.
Internal ear imperfect.
Nose absent.
Nose imperfect.
Nose resembling a proboscis.

OF THE VASCULAR SYSTEM.

Heart absent.

Cavities of heart deficient in number.

a. One auricle and one ventricle.

b. Two auricles and one ventricle.

Septa incomplete.

a. Auricular.

b. Ventricular.

Orifices obstructed or imperfect.

a. Right auricle, ventricular

aperture.

b. Pulmonic aperture.

c. Left auriculo-ventricular

aperture.

d. Aortic aperture.

Foramen ovale permanently closed.

Orifices of aorta and pulmonary

artery transferred.

Orifices of ascending aorta from

left ventricle and of descending

aorta from right ventricle through

the ductus arteriosus.

Commencement of descending

aorta contracted or obliterated.

Foramen ovale persistent.

Ductus arteriosus pervious.

Cardiac valves imperfect.

Pericardium absent.

OF THE RESPIRATORY SYSTEM.

Lung (one or both) absent.

Pulmonary lobes deficient in

number.

Larynx and trachea absent or

imperfect.

OF THE DIGESTIVE SYSTEM.

Œsophagus impervious.

Intestine impervious, or deficient

in various regions.

Anus impervious.

Anus in unusual situations.

Liver preternaturally small.
Gall bladder absent.
Biliary ducts impervious.
Urachus patent. Vitelline duct patent.

OF THE URINARY SYSTEM.

Kidney (one or both) absent.
Kidney lobulated.
Ureters absent or impervious.
Urachus persistent.

OF THE MALE ORGANS OF GENERATION.

Penis diminutive, resembling clitoris.
Prepuce abbreviated—elongated.
Testis (one or both) absent.
External organs absent.

OF THE FEMALE ORGANS OF GENERATION.

Ovary (one or both) absent.
Uterus absent.
Vagina absent.
Vagina impervious.
Vagina a cul de sac.
External organs absent.

MALFORMATIONS RESULTING FROM INCOMPLETE COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD BECOME JOINED.

A.—ON THE ANTERIOR MEDIAN PLANE.

Fissure of the face.
" iris. Coloboma.
" lip.
a. Single harelip.
b. Double harelip.

* To be returned amongst the parasitic diseases of the skin.

Fissure of the palate. <i>a.</i> Hard palate. <i>b.</i> Soft palate. " nose. Naso-buccal fissure. " sternum. " diaphragm. " abdominal walls. " pubic symphysis. " anterior wall of urinary bladder (with extroversion of posterior half). Epispadic fissure of the urethra. Hypospadic fissure of the urethra. Fissure of the scrotum.	Fingers or toes conjoined. Monoculus. Cyclops. Double kidney. MALFORMATIONS RESULTING FROM THE EXTENSION OF A COMMISSURE BETWEEN THE LATERAL HALVES OF PARTS (CAUSING APPARENT DUPLICATION). Double uterus. Double vagina. MALFORMATIONS RESULTING FROM REPETITION OR DUPLICATION OF PARTS IN A SINGLE FÆTUS. Supernumerary fingers and toes. " cavities to heart. " valves. MALFORMATIONS RESULTING FROM THE COALESCENCE OF TWO FÆTUSES, OR OF THEIR PARTS. Fœtus, more or less perfect, contained within another fœtus. Fœtus, more or less perfect, constituting a tumor covered by integument. Double fœtus. <i>a.</i> One perfect. The other an appendage. <i>b.</i> Both more or less perfect. 1. The middle parts united. The upper and lower distinct. 2. The upper parts united. The lower distinct.	3. The lower parts united. The upper distinct. CONGENITAL DISPLACEMENTS AND UNUSUAL POSITIONS OF PARTS OF THE FÆTUS. Transposition of viscera. Hernia or ectopia of the— brain. heart. lungs. intestines. <i>Varieties:</i> Through diaphragm. <i>Syn.</i>, Diaphragmatic hernia. Through abdominal walls. <i>Syn.</i>, Abdominal hernia. Through umbilicus. <i>Syn.</i>, Umbilical hernia. Extroversion of posterior wall of bladder. Testis retained in abdomen. Testis retained in inguinal canal. DISEASES MANIFESTED AT OR AFTER BIRTH. Prematurely born. Stillborn—Asphyxia. Atelectasis pulmonum. Jaundice. Idiotcy. Dumbness or deaf-dumbness. Congenital cataract. Cephalhæmatoma. Syphilis.
B.—ON THE POSTERIOR MEDIAN PLANE. Fissure of the skull. " spinal column. <i>a.</i> Complete. <i>b.</i> Partial. 1. Cervical region. 2. Lumbar " 3. Sacral " Fissure of the spinal cord. MALFORMATION RESULTING FROM COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD REMAIN DISTINCT. Lower extremities conjoined. Syreniform fœtus.		

THE SCIENCE AND PRACTICE OF MEDICINE.

PART III.

THE NATURE OF DISEASES—SPECIAL PATHOLOGY AND THERAPEUTICS.

It is the object of this part to treat of diseases in groups or classes, which possess certain characters or types common to the diseases composing each group; to describe, *Firstly*, The common properties or characters peculiar to the respective classes mentioned in the previous part on systematic medicine; to describe, *Secondly*, The several orders into which these classes of diseases may be subdivided; and, *Thirdly*, To describe in detail the several diseases individually, their *general nature and causes; symptoms, course, and complications; diagnosis, prognosis, and treatment.*

CLASS I.

ZYMOTIC DISEASES.

CHAPTER I.

GENERAL REMARKS ON THE PATHOLOGY OF ZYMOTIC DISEASES.

THIS class comprises diseases which have been observed to be *epidemic, endemic, and contagious*, and includes *specific fevers, small-pox, plague, influenza, cholera*, and such other diseases as possess the peculiar character in common of suddenly attacking

great numbers of people, at intervals, in unfavourable sanitary conditions. In the language of Dr. Farr, the “diseases of this class distinguish one country from another,—one year from another; they have formed epochs in chronology; and, as Niebuhr has shown, have influenced not only the fall of cities, such as Athens and Florence, but of empires; they decimate armies, disable fleets; they take the lives of criminals that justice has not condemned; they redouble the dangers of crowded hospitals; they infest the habitations of the poor, and strike the artizan in his strength down from comfort into helpless poverty; they carry away the infant from the mother’s breast, and the old man at the end of life; but their direst eruptions are excessively fatal to men in the prime and vigour of age. They are emphatically the *morbi populares*.”

The name *Zymotic* (first suggested by Dr. William Farr to designate the class) is not to be understood as implying the hypothesis that these diseases are fermentations, which the derivation of the term would lead one to believe. It has become extensively used of late as applied to the diseases whose characters as a class are already indicated, and for which some convenient term is required. The class, then, to which the term ZYMOTIC has been applied is intended to comprehend all the principal diseases which have prevailed as *epidemics* or *endemics*—all those which are due to *paludal* or *animal malaria*; and those due to *specific disease poisons*, capable of propagation from one human being to another, and *communicable either by direct contact*, or indirectly through various channels of human intercourse, contaminating drinking-water or infecting the air, *or by animals in a state of disease*. The class also comprehends the diseases that result from the *scarcity and the deterioration of the necessary kinds of food*, or from the generation, propagation, or existence of *parasitic animals*. The diseases of this class are thus conveniently arranged into four orders or groups, of which *specific fevers*, *syphilis*, *scurvy*, *worms*, are the common names typical of diseases in the respective groups.

In the greater number of the diseases of this class the blood is more or less changed, and by some is presumed to be the primary seat of diseases which result from specific poisons, of organic origin, either derived from without or generated within the body. These specific poisons tend to produce in the blood an excess of

those decomposing organic compounds which physiology teaches us are always present in the circulating current.

The Physiological Modes in which Poisons act Illustrate by Analogy the Zymotic Diseases.—If the reader will now consider the following statements as to the modes in which poisons act physiologically, he will be prepared to appreciate the effects of those conditions which, like poisons, induce diseases of the class termed Zymotic. The actions of poisons are subject to certain general laws,—the most important of which are, *first*, that they have all certain definite and specific actions; *second*, that they all lie latent in the system a certain but varying period of time before those actions are set up; and *third*, that the phenomena resulting from their action vary, in some degree, according to the dose and to the receptivity of the patient. These laws are common to all poisons, but some are peculiar to individual poisons or classes of poisons, and it may be necessary to notice a few of them.

The *first* law, or that of the definite and specific actions of poisons, cannot be doubted; for if it be supposed that agents acting on the human body do not produce their effects according to certain definite laws, we can neither determine the seat nor the course of any disease, nor direct nor judge of the operation of remedies. No one, for instance, has seen castor oil produce tetanus, or colchicum intoxicate the brain, or opium inflame the spleen. The physician perfectly well knows that the first of these substances acts on the intestines, the second on the ligaments, and the third on the nervous system generally. The action of poisons, therefore, is not accidental, but determined by certain definite laws.

The action of poisons, though definite, is variously limited. Some poisons, for instance, act on one membrane, or on one organ, or on one system of organs; while other poisons extend their action over two or more membranes, or organs, or systems of organs, or even over the whole animal frame. We have examples in aloes and jalap of substances that act mainly upon the mucous membrane of the alimentary canal. In digitalis we have an instance of a medicine that principally acts on the heart, greatly reducing or even stopping its action; while strychnine is an example of a medicine acting on the parts supplied by the spinal cord, producing powerful and sometimes fatal tetanic action of every voluntary muscle in the body.

It is seldom, however, that the action of poisons is limited to one membrane, or organ, or system of organs. The greater number of these noxious agents more usually act on two or more membranes, or organs, or systems of organs. Elaterium, for instance, acts on the mucous membrane of the intestinal canal, and on the kidneys. Tobacco nauseates the stomach, intoxicates the brain, and affects the action of the heart. Antimony has an equally extensive range: it induces cutaneous perspiration, acts cathartically and emetically, and in large doses appears to cause gangrene of the lungs. Alcohol and opium are examples of substances acting still more generally, affecting not only the action or secretion of every organ or tissue of the body, but even in some instances altering their structure. Thus alcohol, in its most limited action, has been shown to cause structural disease of the liver, of the stomach, and of the coats of the arteries. From the circumstance of these substances acting not only generally, but inducing local lesion, they resemble in their effects those of many morbid poisons, as that of typhus fever, of scarlet fever, of small-pox, or of syphilis.

The *second* important law of poisons is, that they lie latent in the system for a period of time which varies in different individuals, before they set up their specific actions. Rhubarb, for instance, produces no immediate result, but lies dormant in the system six or eight hours before its action is sensible on the bowels; opium, in the usual dose, is generally thirty minutes before it subdues the brain. The convulsions from strychnine do not follow till twenty minutes after its administration; and perhaps every substance, except hydrocyanic acid, has a greater or less sensible period of latency.

When a medicine acts on more parts than one, a considerable space of time may elapse after it has affected one organ before it affects another: thus digitalis frequently occasions emesis before it acts on the heart, and the action of mercury on the bowels is frequently sensible for many weeks before the gums and salivary glands are affected. The doctrine of the latency of poisons is indeed so generally admitted that the actual period has been a point on which the condemnation or acquittal of a prisoner tried for murder has turned in our courts of justice, when certain poisons have been supposed to have been given.

The *third* great law of poisons is, that their effects are modified

by the dose, the temperament, and the existing state of the constitution, mentally and bodily, of the recipient. The effect of the dose in modifying the pathological phenomena of disease may be exemplified in the actions of oxalic acid and of arsenic. The specific action of oxalic acid is to inflame the mucous membrane of the stomach ; but to insure this effect the dose must be limited so that this poison may lie in the system many hours. On the contrary, if the dose be excessive, and rapidly absorbed, the poison so disorders all the functions of the three great nervous centres that life is destroyed in a few minutes. Arsenic, likewise, is a poison which inflames and ulcerates the mucous membrane of the alimentary canal, but it requires some hours to set up its specific actions ; for, when the dose is large, it, in like manner, destroys by general irritation, and before traces of morbid change of structure can be appreciated after death. It follows from this law, that the larger the dose, or the greater the intensity of the poison, the more rapid its action, and the less the probability of finding any trace of specific lesion induced by it.

In studying the effects of dose on the constitution, we find some poisons are absorbed and are *cumulative*, while others are not absorbed into the system ; or they are so rapidly removed that no cumulative effect is produced. Thus, in persons predisposed to the effects of digitalis, a dose so small as to produce no sensible effect whatever, will, if frequently repeated, at last destroy the heart's action. This cumulative property of poisons, however, is by no means universal. There is no instance of jalap or of castor oil proving cumulative ; and if a frequent repetition of either of them produces an increased effect, it is, perhaps, in consequence of the nervous papillæ with which they are brought in contact being more easily irritated by each application, and hence they induce a more violent result. That the habitual ingestion of decomposing matter in the water used as drink is capable of inducing conditions favourable to the development of Zymotic diseases, admits of no doubt. Cogent instances of this are to be found recorded in the bitter experience of epidemics of cholera.

Temperament is a circumstance which greatly influences the action of poisons. There are a few persons—rare exceptions—altogether insensible to the action of mercury, so that no quantity will affect their gums, or increase the secretion of the salivary glands. There are others, in like manner, the action

of whose heart no quantity of digitalis will control. On the contrary, there are some constitutions—and these not so rare—so morbidly susceptible of these remedies that it is scarcely possible to administer even a fractional dose of these drugs without giving rise to their specific effects.

Besides natural temperament, *habit*, which may be termed an artificial temperament, has a powerful influence in reconciling us to particular classes of poisons, and of making them even sources of enjoyment. Thus tobacco, alcohol, opium, are all substances which are productive, in the first instance, to many persons, of great discomfort; but by frequent repetition they cease to have any unpleasant effects, and their stimulus at length becomes a necessary indulgence. Still there are many poisons to which no repetition can habituate us. On the contrary, each repetition only the more debilitates the constitution, and renders it more susceptible of the action of the poison.

A peculiar existing state of the constitution has also a powerful influence on the action of poisons; and it would seem proved, with some exceptions, that these agents act with an intensity proportioned to the debilitated state of the patient. There is indeed no duty more imperative on the physician than that of adjusting the dose to the strength of the patient; and nothing is more common than to forbear administering a medicine because the patient's strength will not admit of it. As a general principle, therefore, medicines or poisons may be said to act with a power proportionate to the debility of the patient.

Still there are states of disease which render the constitution of the patient, though greatly debilitated, insusceptible to the action of even powerful remedies. Thus, in typhus fever, the patient will often bear a considerable quantity of vinous stimuli without being affected by it. In tetanus, or hydrophobia, no quantity of opium will tranquillize the symptoms or procure sleep. Fallopius mentions a singular instance of the constitution being armed against the action of a poison. He states that in his day a criminal was given up to himself and other anatomists, to be put to death in any manner they might think proper. To this man, therefore, they administered two drachms of opium, but, labouring under a quartan ague, and the fit just coming on, the "opium was hindered of its effect." The man, therefore, having survived this dose, begged that he might take a similar

quantity, earnestly entreating, if he escaped, that he might be pardoned. The same dose was repeated, but it was in the interval of the attacks; and the man died.

The experiments of Majendie may be referred to as affording many curious proofs of the state of the constitution in accelerating or retarding the action of poisons. He has shown that if a poison be introduced into the system, of such potency as usually will destroy life in two minutes, on bleeding the animal the same result will follow in half a minute, or in one-fourth of the time; and this experiment has often been repeated. Majendie also brought to light the curious fact, that if, after having poisoned the animal, and even after the poison has begun to act, we inject an aqueous fluid into its veins in such quantity as to cause an artificial plethora, as long as this artificial plethora can be maintained, the action of the poison is superseded. No sooner, however, does the plethora cease, from the general effusion of fluid into every cavity of the body, than the poison acts in the usual time, and with even perhaps more than its accustomed severity.

Mr. Hunter thought that no two poisons could *co-exist* in the same system together, or that, co-existing, they could not set up their specific actions at the same time. This hypothesis, however, is unquestionably erroneous; for we constantly see opium and digitalis, jalap and mercury, as well as many other combinations of medicines, producing their respective effects in the same system, and at the same time, by accelerating or retarding each other's actions. There is no truth better established in medicine than that a combination of salts and senna produces a much more efficient and pleasant action than the administration of either remedy separately; and opium is an agent possessing a modifying or controlling power over every organ or tissue, without which it would be impossible, on many occasions, to reconcile the system to the introduction of many necessary and essential remedies. Poisons, therefore, are capable of co-existing together, and of so influencing the system that they reciprocally accelerate or retard each other's actions. The co-existence of two or more specific diseases has been already noticed at page 137.

The general laws observable in the actions of medicinal substances are for the most part precisely similar to those which govern morbid poisons, or only differ in a few minor points; for these latter poisons have their specific actions and their periods

of latency, while their phenomena are not less variable, although the conditions of their varied actions are not yet clearly determined.

The Specific Action of Poisons which Produce Zymotic Diseases is distinctly proved by the fact that we are enabled to determine, within certain limits, the course, symptoms, and pathological phenomena which result from the presence of any given morbid poison. No man, for instance, can confound the phenomena of *small-pox* with those of *intermittent fever*, or those of *intermittent fever* with *syphilis*, or those of *syphilis* with *cholera*: each of these poisons has its separate and peculiar origin, course, development, and mode of propagation, and consequently their actions are so far definite and specific.

The actions of morbid poisons, like those of medicinal substances, are variously limited, some affecting only one membrane, or organ, or system of organs. Thus, *tinea* is an example of a noxious germ acting on one tissue of the body, and even then partially. In some parts of the world, for instance, in Switzerland, in the Brazils, in the Andes, and some of the North-west provinces of India, a poison exists, associated with limestone and sometimes magnesian geological formation, whose action is limited to the undue ossification and thickening of the base of the cranium, tending to diminish the size of the foramina for the blood-vessels, and so leading to cretinism, and to growth of the thyroid gland in goitre (KOLLIKER and REVIEWER in *B. and F. Med.-Chir. Review*, 1861, p. 43). Mr. Ceely, mentioning the fact that at Aylesbury, where goitre prevails, the soil is mainly limestone, incidentally states that solid aggregations of calcareous particles are also found in the thyroid gland. The contagion of *whooping-cough* and the virus of *hydrophobia* affect all the organs supplied by the eighth pair, or pneumogastric system. Instances of morbid poisons acting on several membranes, or organs, or systems of organs, are still more common, and form the great body of this class of diseases. The poison of *measles*, for instance, expresses itself no less on the mucous membrane of the eyes, nose, fauces, and perhaps on the mucous membranes generally, than on the skin; that of *scarlatina* not only on the mucous membrane of the fauces, and on the skin and the kidneys, but also on the serous membranes of the joints and the abdomen. The *paludal* and the *syphilitic poisons* have a still more extensive range, hardly any

organ or tissue of the body being exempt from the destructive ravages of these poisons.

Morbid, like other poisons, have their periods of latency; and, generally speaking, a much longer time elapses before their specific actions come into operation than with medicinal substances. The virus of the *natural small-pox* lies dormant from sixteen to twenty days before it produces any constitutional disturbance; and a still further period elapses, of three or four days, before the specific eruption appears on the skin. The poison of *scarlatina* lies latent from seven to ten days after exposure to the contagion; that of *measles* from ten to fourteen; while the poison of *paludal fever* has been said to lie dormant for a twelvemonth, and that of *hydrophobia* for a still longer time. These are examples of periods of latency far beyond anything that has been observed in the action of medicinal substances; and *syphilis*, in its remote effects upon the organs and the constitution generally, is still more remarkable.

When morbid poisons act on more tissues or organs than one, their actions are sometimes simultaneous, but more commonly they are consecutive, and frequently long intervals of time elapse between each successive attack. Thus, the poison of *typhus* and *enteric fever* may affect the lungs, the membranes of the brain, and the mucous membrane of the alimentary canal, and all these may be attacked contemporaneously, but more often consecutively; or first on the alimentary canal, then on the brain, and lastly on the lungs, several days elapsing between each successive affection.

It occasionally happens that morbid poisons which usually act on a plurality of membranes, exhaust themselves on one or more without affecting others. In *scarlatina simplex* the poison sometimes exhausts itself entirely on the skin, without affecting either the mucous or serous membranes of the body. The *rubeola sine catarrho* is a similar example of the poison exhausting itself on the skin. In *intermittent fever*, when the dose of the poison is limited, and the disease properly treated, it is seldom that a lesion occurs in any organ or tissue; yet, left to run a slow course, with constant exposure to the poison, scarcely any organ or tissue would escape being affected and its function impaired.

Sometimes, when the morbid poison acts on many membranes, the usual order of attack is inverted. In *scarlet fever* the

affection of the skin may precede that of the throat, or the reverse may take place.

It has been seen that the period of latency of medicinal substances having passed over, the effects vary in a considerable degree, according to the dose, temperament, or present state of the constitution of the patient. With respect to the dose of a morbid poison, we rarely possess any direct measure of its strength. The *paludal poison* of tropical climates, to which *malarious fevers* are due, unquestionably greatly exceeds in intensity that of more temperate climates, and its effects are proportionally marked. Thus, in the West Indies, the severe remittent fevers occur with hardly a trace of organic lesion after death, so rapid is their course; in Holland, a paludal fever of less severity exists, but it is followed by enlarged livers or spleens, or by dropsy; while, in this country, the same fever is comparatively mild, and, if properly treated, for the most part terminates without any visceral affection. With respect to the influence of temperament in modifying disease, *small-pox* offers very striking instances. Different persons inoculated or poisoned from the same source have suffered in every degree from this formidable malady—from the *horn*, the *distinct*, the *confluent*, and the *bloody small-pox*; while, in the worst cases, children have died in the primary fever, and even before the specific action on the skin had time to be developed. It may therefore be laid down as a general law, that the more intense the dose of the morbid poison, the more severe the form and rapid the course of disease; and that fewer traces of organic alteration will be found after death when the poison is severe and abundant, than when the poison, or the disorder it produces, has been of a milder character, and the course of the disease more prolonged. Thus, enlarged livers, disorganized spleens, and dropsy, marked every case that died of the Wakheren fever; while in the West Indian and African fevers, though resulting from the same poison, scarcely a trace of disease was to be found.

The existing state of the constitution also influences the event. Thus, persons of a good constitution, but ignorant of their danger, are often seen to pass through a mild form of typhus fever, while the nurses and others contaminated by the same poison, but more alive to their critical state, have sunk in a short time. A presentiment of death is a very unfavourable

circumstance in the progress of *remittent* fever, especially in tropical climates. A soldier will sometimes say to the medical officer, "You have been very kind to me, sir; but this time I shall not get over it." There may be no appearance of absolute or immediate danger at the time—yet the man generally dies (SIR RANALD MARTIN). As a general principle, therefore, it may be stated that morbid poisons act with an intensity proportioned to the enfeebled or depressed state of the constitution; but this law is not universal. Want of a sufficient amount of food is most powerful among the conditions which predispose or help to bring about Zymotic diseases, and most constant in operation. It is a popular belief that the lowering of all the vital forces by deficiency of food constitutes the particular condition which renders a starved population so peculiarly open to the invasion of these diseases; but it is also a curious phenomenon of starvation that a state of general putrescence supervenes during life, as if the want of material for the generation of new tissue were an obstacle to the deportation of that which has become effete (CARPENTER). The hardy mountaineer is a surer victim, whether he visits the low countries of the tropics or the marshes of a more temperate climate, than the feebler native of those countries. The immunity the latter enjoys is probably owing to his habit of living in the noxious atmosphere; for let him remove to a more healthy climate, and then return to those regions of pestilence, and he will be found as susceptible of the poison as the hardier stranger.

Peculiarities in the action of Poisons which induce Zymotic Diseases.

—The principal points in which the effects of poisons which induce Zymotic diseases agree with those of poisons generally having been stated, it will now be necessary to state those circumstances in which they principally differ. Many medicinal poisons have the property of accumulating in the system, and acting with an intensity proportioned, not to the last dose, but to the aggregate of the whole quantity that has been administered. Thus the last few minims of digitalis may stop the action of the heart, or the last few grains of mercury salivate the patient, or the last minute dose of strychnine become fatal. There is, however, no well-authenticated fact which can be arranged under this law in the whole circle of morbid poisons, except, perhaps, the cumulative and persistent pernicious action of paludal malaria.

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The actual quantity required to establish disease, according to the experiments of Dr. Fordyce, is probably extremely small. That physician, in the hopes of mitigating the small-pox, inoculated with virus greatly diluted; and although the disease was not always produced, yet when produced, it assumed every form, character, and degree of severity that small-pox has ever been known to assume.

The puerperal female is not only highly susceptible of poisons of the Zymotic kind, but she is proved to favour their further development; and forms of puerperal fever seem capable of generation by *materies morbi* of a kind other than that which might be considered peculiar to it. It is a well-known fact, unhappily not of rare occurrence, that a medical practitioner or a nurse from a case of puerperal fever going to attend on other cases of labour, the chances are that these will be attacked with the disease. Further, the practitioner or nurse may go to cases of labour from attendance on a case of scarlatina, typhus, erysipelas, or small-pox, and the parturient patients may then become the victims of puerperal fever. In the Vienna Lying-in Hospital it is on record that a mortality of 400 to 500 in an average of 3,000 deliveries *per annum* appeared traceable to the introduction of cadaveric matters, through the uncleanness of the attending students; these matters being especially potent when derived from the bodies of those who have died from the adynamic forms of Zymotic disease. Students of practical midwifery should bear in mind this fact. They ought not to attend cases of labour while they are also engaged with practical anatomy in the dissecting room.

Another peculiar law of morbid poisons, and one wholly unknown in medicinal substances, is the faculty which the human body possesses of generating to an immense extent a poison of the same nature as that by which the disease was originally produced. A quantity of small-pox matter not so big as a pin's head will produce many thousand pustules, each containing fifty times as much pestilent matter as was originally inserted; and, moreover, the blood and all the secretions of the body are equally infected with the matter of the pustules. The miasmata from one child labouring under whooping-cough are sufficient to infect a whole city.

There is still perhaps a more remarkable law of morbid poisons, which is, that many of them possess the extraordinary property of exhausting the constitution of all susceptibility to a second action of the same poison. This is the case with syphilis, scarlatina, measles, typhus fever, small-pox, whooping-cough, and, indeed, with a considerable number of others. Still it would seem that a temporary protective influence is imparted by most morbid poisons, for it is certain that few persons suffer a second attack of the same specific epidemic disease; and, consequently, it follows that the previous action of the poison must for a time impair the susceptibility of the constitution to its attacks. This beneficent law is of great importance in social life; it enables those that have recovered to attend on those that are sick, and allows a mother fearlessly to nurse her child in a dangerous and contagious distemper she has herself passed through, if such an inducement is ever necessary to strengthen the moral courage of a mother.

The laws of poisons are not more important than their *modus operandi*; and this part of the subject has been deeply investigated by modern physiologists, and deserves some consideration. The great and striking alterations which often take place in the blood, led from a very remote period to the doctrine of humoralism, or, that a morbid state of the fluids was the great and primary cause of disease. On the contrary, when anatomy began to be cultivated, and nerves traced into every organ and tissue, it was supposed that disordered actions of these prime agents of motion, and of the great phenomena of animal life, were the great causes of disease, the morbid state of the fluids being secondary. Fontana, attempting to prove this latter theory, found, to his surprise, on laying bare the sciatic nerve in a great number of rabbits, that neither the venom of the viper nor hydrocyanic acid, when applied to it, produced the phenomena of poisoning, and that no other consequence resulted beyond what would have been produced by a similar mechanical injury. Having thus shown that the phenomena of poisoning do not result from the application of the deleterious agent to the trunk of the nerve or to the *solids*, he determined to ascertain whether they followed after absorption, and consequent contamination of the *fluids*. He injected the venom of the viper, hydrocyanic acid, and other poisonous substances

directly into the veins of different animals; and he found that although the nerves of a part may be steeped in these poisons with impunity, yet no sooner did the substance enter the veins than the animal, after uttering a few horrible shrieks, struggled and almost instantly died, and thus demonstrated a morbid state of the fluids, as well as the existence of a tissue of extreme sensibility, with which the poison being brought into contact, accounted for the death of the animal. Fontana pursued this subject one step further, and showed, if poisons acted by absorption, that this absorption was in many instances extremely rapid. He submitted a number of pigeons to be bitten in the leg by a viper. He then chopped the wounded limb off at different intervals after the introduction of the venom, and found, as the result of an extensive series of experiments on several dozens of pigeons, that none recovered when the poisoned leg was removed at a later period than twenty-five seconds, though the phenomena of poisoning did not occur till several minutes later.

The experiments of Fontana had shown (supposing a poison to be introduced into the veins) that all the phenomena of poisoning were accounted for; but still it might be said that to prove the fact of absorption something was wanting in strict demonstration. For the further prosecution of this subject we are indebted to Segalas, who showed that if the arteries and veins of the mesentery of a dog be tied, a quick-acting poison would lie in harmless contact with the corresponding portion of the intestine for many hours; but no sooner were these ligatures removed than poisoning took place in a few minutes. Majendie has carried this proof, of the veins absorbing, even still further. He amputated the leg of a dog, having first introduced a portion of quill into the femoral artery and vein, in such a manner that, on dividing these vessels, the leg hung connected with the trunk solely by means of the quill, all continuity by means of the solids being cut off. The poison was now introduced into the tissues of the paw, and in four minutes the animal was under its influence.

By these experiments it is believed that Fontana, Segalas, and Majendie have completely demonstrated the absorption of poisons by the veins, and consequently of their circulating with the blood; and that no doubt may remain on the subject, modern chemistry has demonstrated the actual presence of many medicinal substances either in the blood itself or in the secretions formed

from it. Thus, after the free use of soda, large quantities of uncombined alkali have been found in the serum. Alcohol has been obtained by distillation from the blood; while iodine, rhubarb, the nitrate of potash, and a large number of other substances taken into the stomach have been found in the urine. It follows, then, that poisons are absorbed and mingled with the blood, and are conveyed directly to the parts on which they act, passing with impunity over others for which they have no affinity.

The fact of morbid poisons in like manner being absorbed, and mingling with the blood, has been shown by many continental writers; but perhaps the experiment made by Professor Coleman is the most satisfactory. "I have produced the disease (the glanders) by first removing the healthy blood from an ass, until the animal was nearly exhausted, and then transfusing from a glandered horse blood from the carotid artery into the jugular vein. The glanders in the ass was rapid in its progress, violent in degree, and from this animal I afterwards produced both glanders and farcy." Scarlatina, measles, and syphilis have now been produced by inoculation *from the blood* of patients labouring under these diseases.

The circumstance of the presence of a poison in the blood is supposed by Andral to produce, besides its toxic states, certain alterations in its physical condition. Thus a specific cause has a tendency to destroy or reduce the quantity of fibrine in the blood, which he has found in some instances to be only one part in a thousand. Hence he adds, whatever may be the nature of the specific pyrexia, the blood always exhibits the following characters, whether it be taken from a vein or collected from the heart and arteries after death,—namely, that the serum and clot are incompletely separated the one from the other, so that the clot is consequently large, and often appears to fill almost entirely the bleeding-basin. Its edges are never raised, and its consistence is inconsiderable, so that it is easily torn, broken down, and reduced to a state of diffuence: in this state it becomes grumous, and discolours the serum. It is also remarkable for the absence of all buff, which is rarely met with in typhus, in measles, in scarlatina, or in small-pox, unless there has been some inflammatory complication; and even when it does exist, as in confluent small-pox, with large collections

of pus, the buff is soft and gelatinous, and, by expression of the serum, is easily reduced to a thin pellicle. This defect of fibrine he conceives to be the cause of the great tendency to hæmorrhage, and to that stasis or congestion so remarkable in typhus fever, scarlatina, and other diseases dependent on morbid poisons.

The facts and arguments which have been adduced prove that morbid poisons act in all instances not capriciously, but according to certain definite and specific laws, modified by the influence of climate, temperament, or the magnitude of the dose: also, that they mingle with the blood, with which they continue in latent combination a certain but varying period of time; and likewise that many of them are capable of co-existing together in the same system. A knowledge of these facts is necessary to the proper understanding of this class of diseases, and it is hoped that by their application many of the difficulties which have hitherto obscured the doctrines of fever, of syphilis, of hydrophobia, and of many other diseases incident to this class of morbid poisons, may be removed, and that this portion of medical science may be placed on a surer foundation, if not on a permanent basis.

Deaths from Zymotic Diseases.—The average annual rate *per cent.* of mortality in Great Britain for the past seventeen years is represented by 2.245; *i. e.*, nearly 22 per 1,000, or 1 in 45 of the population. This statement is given as a fact by which the student may compare the numerical statements which are made in estimating the fatal nature of individual diseases, or of diseases considered in classes.

With regard to diseases of the Zymotic class, it may be stated generally, that from 21 to 26 *per cent.* of the total number of deaths which take place in Great Britain during a year are due to diseases of this class. Generally speaking, also, they may be arranged in the order of their greatest fatality, as follows, namely:—(1.) Cholera, typhus, and other forms of continued fever; (2.) Scarlatina, whooping-cough, measles, croup, small-pox, dysentery, erysipelas; (3.) The other diseases of this class are less fatal; and it has been observed that of late years small-pox, influenza, and typhus fever are less fatal than they used to be.

Under the class of Zymotic diseases the following orders are to be distinguished and described, namely:—

ORDER 1. MIASMATIC DISEASES—*Miasmatici*.

ORDER 2. ENTHETIC DISEASES—*Enthetici*.

ORDER 3. DIETIC DISEASES—*Dietici*.

ORDER 4. PARASITIC DISEASES—*Parasitici*.

CHAPTER II.

PATHOLOGY OF THE MIASMATIC ORDER OF ZYMOTIC DISEASES.

THE diseases to be described under this order acknowledge at least three sources or modes of origin ; while they are all mainly propagated, disseminated, communicated, or diffused through the agency of contaminated persons, food, water, or other agents, or through infected air. The poisons, miasms, gases, germs, active principles, or morbid agents may be arranged under three classes, namely,—1st. Paludal malarious poison ; 2d. Animal malaria poison ; 3d. Specific disease poisons. The diseases they engender are attended by a febrile state which may assume various forms or types.

Paludal Malarious Poison.—This poison arises from marshy land in particular conditions, such as decomposition, under the influence of partial moisture, and of heat above 60° Fahr. If the land is perfectly dry or perfectly flooded the poison is not generated. It is a material poison. It may be wafted along with the wind, and so induce fever at a distance from the place where the poison is generated. It may also be intercepted by a belt of trees. It appears to be most intense near the surface of the ground. The diseases usually attributed to this endemic source, and which were formerly so destructive, have almost disappeared from this country. The reason of this may fairly be ascribed to the improved drainage both of the towns and of the agricultural districts. The fact may be proved, did space permit ; and the practical inference leads one to hope for still more immunity from diseases arising from this source, if the “proper authorities” direct further efforts in this direction. “Within the last half-century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and

often injurious in their effects in the first instance, they have become of the first importance. Land has, in many instances, doubled in value; and town-sewering, with other social regulations, have not unfrequently prolonged human life from five to fifty per cent., as compared with previous rates in the same district." "Agues (and malarious cachexiæ) are reduced. Since 1840 an annual mortality in English towns of 44 in 1,000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15; and human life has now more value in England than in any other country in the world—a result entirely due to better sanitary arrangements" (Rawlinson, *Journal of Society of Arts*, March 21, 1862, vol. x., p. 276).

The time, indeed, appears to have arrived when accurate sanitary statistics should not only be kept for all branches of the public service, but also by all corporations, municipalities, boards of commissioners, and parish vestries, for the population within their respective jurisdictions. Such statistics should be published at least once every year, as the natural history of the population (*Sanitary Statistics, B., International Statistical Congress*. London, 1860. Second Section).

Animal Malaria Poisons or Effluvia.—Animal effluvia arise from the decomposition of the exhalations, excrements, or excretions of individuals (whether of mankind or of the brute creation), of filthy habits, or when crowded in confined spaces. Such poisons appear to be more limited in the causation of disease than the paludal poisons just noticed. They are developed in situations where numbers are crowded together, as in prisons, hospitals, besieged towns, camps, ships, and such-like places. Winter is known to be favourable to their development and deleterious influences. They are sedative or depressing in their actions, and while they lower the energies of the nervous system, they tend to corrupt or poison the blood, surcharging it with decomposing organic compounds.

Ephemeral febrile states are produced, and such conditions of ill-health are thereby established, so that a certain proclivity to the specific communicable diseases seems to be entailed. Conditions of the constitution are brought about by which a certain receptivity or disposition of the blood is produced, rendering it more liable to undergo those changes which it undoubtedly undergoes during the progress of the specific diseases about to

be noticed. Thus we have a predisposition to certain diseases brought about, and especially to such specific febrile affections as *typhus fever*, *dysentery*, *cholera*, and the like. Dr. Carpenter (than whom we have not a better physiological authority), in an interesting paper on the "Predisposing Causes of Epidemics," shows that the conditions which tend to bring about the specific miasmatic diseases of the Zymotic class are referable to three categories:—(1.) Conditions which may introduce into the system decomposing matter that has been generated in some external source. (2.) Conditions which occasion an increased production of decomposing matter in the system itself. (3.) Conditions which obstruct the elimination of the decomposing matter normally or excessively generated within the system, or abnormally introduced into it from without.

The decomposing matters generated in external sources may be enumerated as putrescent food, water contaminated by sewerage or other decomposing organic substances, and air charged with miasmatic emanations. The constant breathing of such putrescent effluvia may, by communicating a putrescent tendency to the blood, render it more prone to the changes by which specific poisons are multiplied. Ill-health and consequent receptivity for any specific disease poison are developed by the degeneration of the tissues within the body, such as occur in the puerperal state, after severe injuries, and as a consequence of excessive muscular exertion; and also by an insufficient supply of air, a high external temperature, and the ingestion of alcoholic drinks. Each and all of these causes tend to induce morbid conditions of the blood, a tendency to putrescence, and a condition of ill-health of the body.

Specific Disease Poisons.—The matter by which the specific miasmatic diseases are communicated and propagated is solely derived from the body of the similarly diseased human or animal being; for there, during the course of the specific disease, is the soil in which the specific poison is bred, to multiply and propagate its kind. It is not yet clearly established how far the bodies of animals may not be a soil for the propagation of diseases communicable to man. (See the Sections on "Small-pox" and "Cow-pox.")

The diseases of the lower animals are not sufficiently studied by us. The diseases of plants are almost entirely neglected.

Yet it is clear that until all these have been studied, and some steps taken to generalize them, every conclusion in pathology regarding the nature of the propagation and dissemination of specific miasmatic, and even of parasitic, dietic, and enthetic diseases must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the causes of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food.

Dr. Carter, of Bombay, has shown that there is in India a very singular, and although strictly endemic disease, yet a very prevalent one, which occurs in the hands and feet, especially the latter, and which it is probable is really of the nature of a "blight," in so far as it is owing to the implantation in the tissues of "sporules or germs," which in the progress of development commit irremediable ravages on the affected parts, leading ultimately to entire disorganization of the tissues. It is known as the "fungus disease of India," originally described by Dr. Carter in the *Transactions of the Medical and Physical Society of Bombay*, No. 6, new series for 1860. (See the account of "Parasitic Diseases" at the end of this volume.)

On the relations between the diseases of man and animals, and especially in connection with food, the reader is referred to a series of papers by the author, in the *Medical Times and Gazette* for 1857.

Dr. William Budd, of Bristol, has also recently directed attention to the occurrence of malignant pustule in *England*, in a paper read at the great meeting of the British Medical Association in London, in August, 1862. He has shown that the disease has not been so uncommon in England as had been supposed—that it is common and very fatal to oxen and sheep in this country—that in man and in sheep the disease is identical—that it is communicable to man by direct inoculation, and also by eating the flesh of the animals affected—that it may be conveyed and disseminated by the bites of insects, such as gnats—and that the disease may be re-communicated from man to animals (*Brit. Med. Journal*, January 24, 1863).

There are some peculiar and characteristic features especially

pertaining to the specific diseases of the Miasmatic order which require special notice as introductory to a description of the individual diseases:—First, *They suddenly spring up in a locality—under unfavourable sanitary conditions.* Second, *They may rapidly spread at irregular intervals, so as to incapacitate or destroy great numbers of people.* These two marked and striking features are technically described as being due respectively to *Endemic* and *Epidemic influences*, the nature of which will be considered in the following chapter.*

CHAPTER III.

ON THE NATURE OF ENDEMIC, EPIDEMIC, AND PANDEMIC INFLUENCES.

Endemic Influences result from those conditions or agencies peculiar to a locality which favour the development of various miasmatic diseases, and may thus account for their sudden origin. Such diseases are then said to be *endemic*. These endemic influences, for the most part, are exerted by the geological properties of a district, and are traceable to the constitution and state of the soil, water, and air; to elevation above the level of the sea, vicinity of sea, rivers, or stagnant water, woods, and vegetation; variations of temperature, prevalent winds; in connection with avocations, modes of life, quality of food and quantity, as modified by moral agencies, such as indolence or activity; privation and comforts, filth or cleanliness of people; together with their habits of life and employments, ignorance or mental culture; and lastly, their social, moral, religious, and political conditions. It may be shortly stated in illustration, that endemic influences become mainly active through the following conditions, namely:—1st. That the specific poisons by which the

* Deputy Inspector-General Dr. Lawson has recently called the attention of the profession to what he considers to be *oscillations* of influences over the whole world, determining febrile diseases. To such influences he gives the name of "Pandemic" (*Transactions of Epidemiological Society* for 1862; also, *Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1861).

communicable diseases, such as small-pox or typhoid fever, propagate their kind are never totally in abeyance. 2d. The specific communicable diseases are constantly extant somewhere, and only under conditions favourable to their dissemination do they spread or become epidemic. Although their germs, specific gases, active principles, or media of propagation and development may lie dormant or latent for a time, it is not to be inferred that they have ceased to exist. 3d. The history of all the specific communicable diseases demonstrates the same alternations of slumber and activity; of wide-spread prevalence in one place, while neighbouring places may remain free; and finally, the same successive invasion of neighbouring places, such that the prevailing disease only begins to prevail in the new locality after it has already died out in the old. 4th. One element remains constant in the history of endemic influence, and that is *the specific morbid poison* which is the origin of each case. It is susceptible of transmission from place to place, gathering strength as it proceeds, again to die out or become dormant, so that its track is with difficulty followed or traced out. 5th. In large cities such specific poisons are always more or less active, and their diseases always present; but in the country districts they only now and then occur. The occurrence of long intervals of rural exemption is not traceable to any feebleness of the poison to act; for when the disease does become developed in these places, the ratio of persons or of animals attacked is incomparably greater than is ever seen in cities under like circumstances (see Professor Acland's account of the fever in Great Horwood, in 1857-58; and Dr. William Budd, of Clifton, regarding fever at North Tawton; and his most instructive little book *On the Propagation of Typhoid Fever*). 6th. In large towns the sewers are constantly charged with the *materies morbi* of specific diseases always abounding in towns. In small villages, and other places where no sewers exist, the air only may be infected, or the water contaminated, by the direct or indirect importation of cases of specific disease or their equivalents—the poison itself—so that the organic impurities, the dung-heaps, the open soil which surrounds the dwellings of the patients, the cess-pools, and the privies common to several houses, gradually but eventually become impregnated with the specific poison of the disease. Thus the atmosphere of the village may become incomparably

more virulent than the atmosphere of the sick-chamber itself. Hence the rapid epidemic spread of the miasmatic diseases in the limited space of rural villages; and which gives rise to the popular error, that such diseases are invariably contagious in country places, and only rarely so, or by exception, in cities or large towns. 7th. All these specific diseases multiply their kind after similar modes of propagation. 8th. All of them establish a constant series of morbid changes and lesions, and always issue in the reproduction of its own specific germ, miasm, gas, morbid poison, or active principle by which it propagates its kind. Thus small-pox propagates small-pox; measles multiplies measles; scarlatina reproduces scarlatina; typhoid fever breeds typhoid fever; typhus, typhus; and so on. In the terse language of Dr. William Budd,—“What small-pox and measles were in the Arab in the days of Rhazes, they still are in the London Cockney of our own time. What they are in the London Cockney, they are in the wild Indian of the North American prairie, and in the Negro of the Gold Coast. To all the other specific communicable diseases, as far as our records go, the same remark applies. In races the most diverse, under climates the most various, age after age, through endless generations of man, these diseases pass down through the human body (sometimes through animals—*e. g.*, ovine small-pox?), perpetuating their own kind, and each maintaining its separate identity by marks as specific as those which distinguish the asp from the adder or the hemlock from the poppy.” Such being the case, it is difficult to conceive (as Drs. Watson and William Budd most justly observe), “*that diseases of whose propagation this is the history can ever be generated in any other way.*” Most of these miasmatic diseases also are peculiar to man; while animals on their part are infested by a whole brood of communicable diseases, no less specific in their kind, each distinct from the other, and most of them, although some may be communicable to man, are incapable of multiplying in the human body. Cattle appear to be subject to a variety of malignant and communicable fevers from which man is altogether exempt. 9th. Certain receptive conditions, or a predisposition (the nature of which is unknown), exists in individuals, which appear essential to the development of the specific poisons and the establishment of the disease; and immunity against a repetition of the disease is generally conferred by one attack of the same disease—an

immunity which has been proved by experiment on an enormous scale with regard to small-pox; and with regard to the other diseases of this kind, the belief in such immunity is deduced from extensive observation. But the immunity acquired by one attack of any of these diseases is of no avail against the rest. Measles, for example, renders the body proof against measles, but leaves it as open to small-pox as before, and so on of the rest. 10th. With regard to fermentation, putrescency, or decomposition, there is some reason to believe (as shown in the previous chapter) that it may quicken the activity or facilitate the development of specific morbid poison, in the way of a predisposing cause. Dr. Budd, however, believes that this effect has been much overrated, notwithstanding the observations of Dr. Carpenter referred to in the preceding pages. Nevertheless, there is no small amount of circumstantial evidence tending to show that endemic conditions may be thus far favourable to the propagation of specific diseases, even to the extent of epidemics, in consequence of the predisposing agency of putrescent emanations; and on the other hand, both endemic and epidemic influences are often held in abeyance by the tendency to decay, decomposition, and destruction of the specific germs, miasms, gases, or disease poisons themselves. They are stamped with the tendency to change and to perish. Like all organic substances which propagate from minute or invisible beginnings, myriads perish for one that is fruitful. This is especially demonstrable in respect of the Parasitic diseases, whose germs would overrun the world if they all came to maturity; but the extinction or the dispersion of the specific poisons is abundantly provided for through the operation of many natural causes; and by imitating some of these operations of Nature we may be able eventually to exterminate, or, at all events, greatly to modify the severity and reduce the mortality from many of these diseases.

The belief in the spontaneous endemic origin of the specific miasmatic diseases rests on evidence entirely *negative*—namely, the fact that cases do spring up in which it is impossible to trace the disease back to a personal source of specific propagation and dissemination—an event which is inherent in the very nature of these diseases. For the active principle of the poison is invisible, although the matter that is known to contain it may be capable of isolation and inoculation, as in small-pox; yet the existence of

the specific disease poison is known to us by inference only. Again, we know that ample provision is made and ways are open for the dissemination of the active agent of propagation in a thousand unseen modes, so that it is obvious that the precise source of infection and its track must often baffle the wisdom of man to discover or trace out.

Cases thus constantly arise which appear to give countenance to the belief that the disease has had a spontaneous origin—*sporadic*, as it is termed. Numerous cases of small-pox occur which can never be traced to their source, or to communication with persons similarly diseased; yet the history of small-pox is decisive against the notion of its spontaneous origin; and if of small-pox, so for all the other specific Zymotic diseases of the same nature. Dr. Watson has well observed that “the small-pox never occurs except from contagion. *It was quite unknown in Europe till the beginning of the eighth century.* No mention of any such malady is to be found in the Greek or Roman authors of antiquity. Now, whatever may have been the deficiencies of the ancient physicians, they were excellent observers and capital describers of disease; and it is impossible that a disease so diffusive, and marked by characters so definite and conspicuous, should have escaped their notice, or have been obscurely portrayed (if known) in their writings. On the other hand, Mr. Moore, in his learned and interesting *History of Small-Pox*, has shown that it prevailed in China and Hindostan from a very early period—even more than a thousand years before the time of our Saviour. That it did not sooner extend westward into Persia, and thence into Greece, may be attributed partly to the horror which the disease everywhere inspired, and the attempts that were subsequently made to check its progress, by prohibiting all communication with the sick, partly to the limited intercourse which then took place among the eastern nations, but principally to the peculiar situation of the regions through which the infection was diffused, separated as they are from the rest of the world by immense deserts and by the ocean” (Watson, *Lectures on the Practice of Physic*, 3d edition, vol. ii., p. 709). “If anything were wanting,” writes Dr. Budd, “to show what is the true inference to be drawn from these events, it would be found in the fact that, *once imported into the West*, it spread with the most fearful rapidity and havoc; and that while almost all men are prone to take the disorder, large portions

of the world have remained for centuries exempt from it, until at length it was imported, and that then it infallibly diffused and established itself in those parts. In this country the (endemic) conditions for the spread of the disease existed in the most intense degree, as was shown by the event when the disease was once introduced. The long lapse of ages during which we remained entirely free from small-pox showed, with equal clearness, that, until this introduction occurred, all the conditions favourable to the development of small-pox were powerless to cause a single case. The spectacle witnessed in Europe was repeated over again in the Western World in a still more striking way. Our knowledge of the events here is precise and sure. There was no small-pox in the New World before its discovery by Columbus, in 1492. In 1517 the disease was imported into St. Domingo. Three years later, in one of the Spanish expeditions from Cuba to Mexico, a Negro covered with the pustules of small-pox was landed on the Mexican coast. *From him* the disease spread with such desolation that within a very short time (according to Robertson) three millions and a half of people were destroyed by it in that kingdom alone." "Again, small-pox was introduced into Iceland in 1707, when sixteen thousand persons were carried off by its ravages—more than a fourth part of the whole population of the island. It reached Greenland still later, appearing there for the first time in 1733, and spreading so fatally as almost to depopulate the island," (Budd, l. c., p. 35, *et seq.*) No common conditions of human life gave rise to such phenomena. Propagation from the actual poison of a pre-existing case was the one necessary and all-sufficient condition for these endemic outbreaks and their epidemic prevalence. The precise mode in which the miasmatic diseases, with their specific poisons, first came into existence is beyond our *ken*—hidden from us as yet by a vail, and remaining an inscrutable, at least an unpenetrated, mystery. But everything tends to show that once created, they all propagate only in one way—namely, by continuous succession.

Defective ventilation, inasmuch as it is always injurious to health, always aggravates disease, and so promotes the endemic influence. With regard to any influence it may have on the development and spread of communicable disease, it may be noticed that it does not equally help *all* communicable diseases to

develop themselves and to spread. Commonly it seems to operate in proportion as the specific miasmatic disease is one which imparts specific poison properties to the general exhalations of the sick. The significance of defective ventilation is not likely, therefore, to be quite the same where typhoid fever, cholera, or dysentery are the prevailing diseases, as where the disease is typhus fever, scarlatina, small-pox, or diphtheria. In the cases of typhoid fever, cholera, and dysentery, any defect of ventilation would become more and more important in proportion as the bowel discharges of the sick were not promptly removed from within doors, or as, from other causes, there were faecal effluvia or excrements suffered to remain in the dwelling (Simon, *Third Report of the Medical Officer of the Privy Council*, 1860, p. 10).

Epidemic Influence.—The second characteristic feature peculiar to some of the miasmatic order of Zymotic diseases is, that they sometimes spread rapidly, so as to incapacitate and destroy great numbers of the people. The disease is then said to be *epidemic* (ἐπι, upon; and δῆμος, the people). No subject has afforded greater scope for speculation than the origin, cause, and progress of epidemics. It is in vain to speculate upon the subject; and, in the words of Dr. Wood, of Pennsylvania, “all we can say, with certainty, regarding epidemics, is, that there must be some distempered condition of the circumstances around us—some secret power that is operating injuriously upon our system—and to this we give the name of *epidemic influence* or *constitution*,” and which is believed to predispose towards the receptivity of specific disease poisons. The observations of Mr. Simon lead to the belief that the prevalence of external conditions, tending in certain localities to determine a specific decomposition of excrement, communicable to other organic substances and infecting the air, is an essential element in an epidemic period.

The most recent speculation regards the discovery of a peculiar atmospheric condition, ascribed to a principle called *ozone*, or *osmazone* (ὀζων, stink, or ὀσμὴ, smell), of which, as yet, we know nothing definite, although many subtle instruments and apparatus are in use to detect and measure the amount of this principle in the air.

A careful study of the effects of the *epidemic influence* appears to warrant the enunciation of certain laws which seem to regulate its operations. These laws may thus be condensed:—

Laws of Epidemic Influence.—(1.) This influence frequently predisposes to diseases, apparently independently of any other known cause, as in the case of influenza and cholera. It makes itself manifest by appearing to give increased energy to causes which produce particular diseases; so that small-pox, scarlatina, typhus, and the like, sometimes rage with great violence as epidemics. It also appears to predispose to new and anomalous forms of disease, as witnessed in the furunculoid epidemic which recently prevailed both in Europe and America, from 1849 till 1852. (2.) Sometimes the *epidemic influence* manifests itself by a certain type or direction which existing diseases appear to take. Thus, at one period diseases take a low, or what is called a *typhoid* type, so that depletion is not tolerated; at another time an *inflammatory* tendency predominates, and antiphlogistic treatment is required. At one period there is a tendency in disease to complicate its course by a disposition to affect particular organs. At one time head affections predominate; at another time affections of the chest, or of the alimentary canal, complicate the course of a prevailing disease. Consequently the same disease may demand very different, and even opposite, modes of *management*. (3.) During epidemics other diseases are apt to assume more or less of the prevailing epidemic features. Thus, when cholera prevails, looseness of the bowels often complicates the course of other affections. When influenza prevails, catarrhal complications increase the danger of other diseases. Ill-health of any kind, therefore, favours the action of the epidemic influence. (4.) Some change in the character of prevailing diseases of a constant and recurring kind often indicates the approach of an epidemic and the prevalence of the epidemic influence. (5.) The first effects of the *epidemic influence* are usually the most violent and marked, and the cases of the epidemic disease become mild as the *epidemic influence* passes away. (6.) The *epidemic influence* sometimes disappears entirely after a short prevalence; sometimes continues, with irregular intermissions, for two, three, four, or even six years, or longer. Influenza and cholera are examples. (7.) An epidemic tendency, after continuing for several years, may give place to one of a different kind, which, in its turn, may again give place to the first. *Malarious fevers*, *yellow fever*, and *typhus* illustrate this in America. The eruptive affections seem to run in somewhat

similar cycles. After the introduction of vaccination the *small-pox* seemed for many years to be almost entirely subdued; but more recently again the disease has seldom been entirely absent from among us, alternating as an epidemic now and then with *measles*, *scarlet fever*, and *typhus*. We look forward to the time when vaccination, enforced by law, will completely eradicate the disease. (8.) The lower animals are also subject to epidemic influences; and seasons of unusual fatality among them have coincided with those in which the human race have suffered. This fact has been well shown in an elaborate and erudite analysis of the census of Ireland, by Sir William R. Wilde, of Dublin, the diseases of the population having been recorded at the time.

Pandemic Influences.—The expressions of the hitherto prevailing doctrines regarding *endemic* and *epidemic* influences appear so unsatisfactory to many minds, and leave many circumstances regarding the spread of diseases unexplained, that attention is being directed to more comprehensive views and investigations of the questions involved in the preceding paragraphs. An ingenious theory has been propounded by Deputy Inspector-General Dr. Lawson, who has attempted to establish the occurrence, between 1817 and 1836, of a series of *oscillations of febrile diseases*, following each other over the world with amazing regularity. The mode of occurrence of such febrile diseases he attributes to a cause or influence which, from its extent and *progressive* character, he names a "*pandemic wave*," to distinguish the influence from that usually understood as *epidemic*, referring to a single form of disease affecting a limited space. Under the influence of this *pandemic wave* Dr. Lawson believes that there is a constantly progressive tendency to the development of all *endemic* febrile diseases in the Atlantic and Western parts of the Indian Ocean, from South or South-east to North or North-east.

But the facts and data on which this theory is made to rest are not of sufficient number, and many of them are not sufficiently trustworthy, to rest a judgment upon. In not a few instances a totally different interpretation may be given to that which Dr. Lawson has assigned to them. Although, therefore, it may be premature to propound such a theory, especially as it is still open to the verdict of "not proven," yet the expression of it is calculated to do good by drawing attention to the subject,

and to the comprehensive, world-wide range which must be given to such investigations; and to whom can Science look with more hope for results than to the medical officers of Her Majesty's British and Indian armies?

A successful study of these peculiar and characteristic features of miasmatic diseases—namely, the *endemic*, *epidemic*, and *pandemic influences*—is of the utmost importance to the student. He will learn to appreciate how much and successfully mortality may be diminished by well-directed hygienic measures, such as cultivation and improvement of the soil, extension of commerce, improvements in diet and the social circumstances of the lower classes—especially in regard to cleanliness, ventilation, and domestic management of improved dwellings, and efficient sewerage; care in the separation and treatment of the sick when in numbers; and the use of strict measures of a prophylactic kind suited to the circumstances of the case. Next to large towns, the health of the Army is of the greatest importance, especially when we consider the tendency that exists to a high rate of mortality in that service. In the military age (which is the age between eighteen and forty) the mortality of the general population in England is less than one per cent. per annum. The mortality of the British army is much above this. On Home service it has had a mortality double that of the civil population at the corresponding ages; and seven-ninths of the entire mortality among the infantry of the line has arisen from diseases of the Zymotic class. Disease and mortality are much greater during campaigns, when more than twenty-two per cent. are constantly on the sick list. The causes of high rates of mortality require constant investigation, by carefully observing, recording, and comparing the facts over a sufficiently large area; thus arriving at certainty as to the causes, and whether they can be mitigated or removed.

An observation of great interest in connection with *animal malaria poison*, as well as with *epidemic influences*, may be appropriately referred to here. It seems clearly proven, especially by the valuable and decisive observations of Dr. William Budd, of Bristol, that the communicable poisons of typhoid fever and of cholera are capable of being imported or carried from place to place by persons who have the disease. Dr. Budd's history of the North Tawton fever and its offshoots (*Lancet*,

July 9, 1860) is most conclusive on this point. His arguments are also cogent to the general effect that *specially* the bowel discharges are *means* by which a patient, whether migrating or stationary, can be instrumental in disseminating typhoid fever and cholera. Mr. Simon makes the important remark, however, that these bowel discharges may not be the *sole means* of multiplying and disseminating these diseases; although, provisionally, the conclusions of Dr. Budd must be acted upon in their present unqualified form; while it is of the greatest practical importance to learn, as exactly as possible, whether it is in all states of the disease, and under all circumstances, that the bowel discharges of typhoid fever and cholera can communicate and multiply the means of dissemination. In illustration of such possible contingent results, Mr. Simon refers to some interesting and important experiments made in 1854 by Professor Thiersch, of Erlangen. These experiments seemed to show that cholera evacuations, *in the course of their decomposition*, either acquire the power of communicating or multiplying their specific poison, or that the specific poison inherent in them becomes intensified by decomposition (Zymosis?). That the decomposition or change may begin even in the bowels, after the secretion and accumulation of the material in them, as well as in cess-pools, seems to be possible; and perhaps, as Mr. Simon justly remarks, may furnish an explanation of the many cases in which human intercourse has apparently disseminated the disease. For, according to the observations of Professor Pettenkofer at Munich, and Professor Acland at Oxford, it would seem that during cholera periods the immigration of persons suffering apparently only from diarrhoea has been followed by outbreaks of cholera in places previously uninfected; and Professor Pettenkofer ascribes this fact to an influence (Zymotic?) exerted by the decomposing fæces of such diseased persons, in the cess-pools and adjoining soil of ill-conditioned places to which they go. Specific-poison-properties of this kind would thus arise, and probably extend to the pollution of well-waters of such soils, and might render them, if swallowed, capable of exciting cholera, or typhoid fever, or dysentery, by direct *contagion*; and so any of these diseases would thus have all the appearance of having arisen *de novo*. It is encouraging to sanitary reformers, as Mr. Simon justly remarks, that cases of the apparent introduction of cholera contagion by

human*intercourse are essentially different from cases of the dissemination of such specific diseases as small-pox or measles. The multiplication of the specific poison in the latter diseases takes place exclusively within the human body. The multiplication and dissemination of them have no immediate dependence on differences of medium; but wherever human beings can cross one another's path, to the susceptible or unprotected person these specific diseases may be communicated. On the other hand, it seems really to be the fact that the cholera poison (and probably, also, typhoid fever poison and dysentery), if it can at all be multiplied within the body, almost certainly has its great centres of multiplication *elsewhere*; namely, in those avoidable foci of corruption where excrement accumulates and decays. Military authorities ought to remember this fact. They have had abundant evidence of it in the old camping grounds of the Indian army, as well as when following the route and encamping on the ground previously occupied by retreating armies. For disseminating the disease and multiplying the poison, foulness of medium seems indispensable; and it is no ordinary foulness which will impart to air, food, or water the Zymotic action of decomposing excrement. The common taint is something specific. Therefore, as regards cholera, it seems highly probable that the immigration of infected persons might occur to a very great extent without exciting epidemic outbreaks, if such immigration were only made into places of irreproachable sanitary conditions, especially as regards water supply and the continuous removal of house refuse or camp filth. (Compare Simon in his public health reports—especially *second* and *third*—relative to the people of England; also Pettenkofer, Acland, and Thiersch, as quoted by Simon, p. 3 of his *third* report, 1860.)

CHAPTER IV.

MANAGEMENT OF EPIDEMICS; AND ON PROCEEDINGS WHICH ARE
ADVISABLE TO BE TAKEN IN PLACES ATTACKED OR THREAT-
ENED BY EPIDEMIC DISEASES.

THE practical questions immediately involved in the exposition which has been given of the nature of Zymotic diseases in

general, and of the miasmatic order of these diseases in particular, is contained in the following statement, namely:—That it is possible to extinguish the greater number of epidemic diseases, however intense or abundant may be the atmospheric or other agencies which constitute their potential causes, by remembering,—(1.) That the living body of the diseased persons is the soil on which the communicable disease breeds the poison by which the specific disease is multiplied and propagated: (2.) That excretions from an infected person, especially such excretions as are immediately related to or flow from parts affected with specific lesions, probably contain the most active elements of the specific poison by which the disease may be disseminated: (3.) That such active elements, germs, poisons, miasms, gases, or noxious agents may contaminate the drinking-waters of a district, or may infect the atmosphere, or lie dormant for variable and unknown periods of time, just as seeds dry up and preserve their vital properties: (4.) To follow out zealously the hygienic measures which flow from these statements, and so prevent the propagation of specific diseases: (5.) To preserve as much as possible the blood of every individual in that state which shall prevent these poisons from finding the conditions of their development within the body: (6.) That these ends are to be attained on the one hand by preventing the production of fermentable matter in or out of the body; and on the other hand by promoting its removal and chemical destruction or decomposition, when it is inevitably generated, and by a free supply of pure air, and by the reduction of that air to the lowest temperature at which the condition of the individuals will allow it to be safely inhaled. Preventive measures based upon these principles are of the utmost importance, so much so that the most eminent members of the medical profession in London and elsewhere concur in the views and opinions of Dr. William Budd, unanimously cherishing the maxim, that, “except under the pressure of great military straits, no army ought ever to suffer on a large scale from this great group of communicable diseases, and especially such as are disseminated by intestinal discharges.”

The following detail of proceedings advisable to be taken in places attacked or threatened by epidemic diseases are given mainly from a memorandum drawn up by John Simon, Esq., the Medical Officer of the Privy Council, and published in his *Third Report on the Public Health in England in 1860*:—

1. Wherever there is prevalence or threatening of cholera, diphtheria, typhus, or any other epidemic disease, it is of more than common importance that the powers conferred by the Nuisances Removal Acts, and by various other laws for the protection of the Public Health, be vigorously, but at the same time judiciously exercised by those in whom they are vested; and with regard to armies, that the instructions relative to the guidance of the Medical Officer in sanitary matters, contained in the *Army Regulations*, be duly carried out, on the principle that the executive should act under authority, in order to carry out the required measures efficiently.

2. If the danger be considerable, it will be expedient that the local authorities in civil life, and the commanding officers of armies, brigades, divisions, and regiments in military life, avail themselves, as soon as possible, of the medical advice within their reach, in taking measures of prevention and protection against the spread of epidemic influences.

3. Measures of precaution for prevention and protection are equally proper for all classes of society, civil and military. But it is chiefly with regard to the poorer civil population—therefore chiefly in the courts and alleys of towns, and at the labourers' cottages of country districts—that local authorities are called upon to exercise the utmost vigilance, and to proffer information and advice. Common lodging-houses, and houses which are sublet in several small holdings, always require particular attention.

4. Wherever there is accumulation, stink, or soakage of house refuse, or of other decaying animal or vegetable matter, the nuisance should as promptly as possible be abated, and precaution should be taken not to let it recur. Especially all complaints which refer to *sewers and drains*, or to *foul ditches* and *ponding of drainage*, or to *neglect of scavenging* should receive immediate attention. The *trapping of house drains and sinks*, and the state of *cess-pools* and *middens*, should be carefully seen to. In *slaughter-houses*, and other places where beasts are kept, strict cleanliness should be enforced.

5. In order to guard against the harm which sometimes arises from disturbing heaps of offensive matter, it is often necessary to combine the use of chemical disinfectants with such means as are taken for the removal of filth; and in cases where removal is for the time impossible or inexpedient, the filth should always be disinfected. Disinfection is likewise desirable for unpaved earth

close to dwellings, if it be sodden with slops and filth. Generally, where cholera or typhoid fever is in a house or barrack, hospital or hut, the privies especially require to be disinfected.

6. Sources of water supply should be carefully and efficiently examined. Those of them which are in any way tainted by animal or vegetable refuse—above all, those into which there is any leakage or filtration from sewers, drains, *cess-pools*, or *foul ditches*—ought no longer to be drunk from. Where the disease is cholera, diarrhœa, or typhoid fever, it is especially essential that no foul water be drunk.

7. The washing and lime-whiting of uncleanly premises (houses, huts, hospitals, barrack guard-rooms, and the like), especially of such as are densely or multifariously occupied, should be pressed with all practicable despatch.

8. Overcrowding should be prevented. Especially where disease has begun, the sick-room should, as far as possible, be free from persons who are not of use or comfort to the patient.

9. Ample ventilation should be enforced. Window frames should be seen to, (1.) That they may be made to open, if not so made; and (2.) That they be kept sufficiently open. Especially where any kind of specific disease, communicable by infection of the air, has begun, it is essential, both for patients and for persons who are about them, that the sick-room and the sick-house or hospital be constantly and efficiently traversed by streams of fresh air. This is especially necessary at night, and steps should be taken to insure efficient ventilation even at some real or imaginary expense of comfort.

10. The cleanest domestic habits should be enjoined. Refuse matters should never be suffered to remain or to linger within the dwelling, hospital, barrack-room, or hut. Such refuse must *at once* be removed, and at once disposed of, or cast into the receptacle provided for it. All things or utensils which have to be disinfected or cleansed should always be disinfected or cleansed *without delay*.

11. With regard to material substances discharged or separated from the bodies of the sick, special precautions of cleanliness and disinfection are necessary. Among discharges or substances separated from the body which it is proper to treat as capable of communicating disease, are those which come, in cases of small-pox, from the affected skin; in cases of cholera and typhoid fever,

from the intestinal canal; in cases of diphtheria and scarlatina maligna, from the nose and throat, and the exhalations from the skin and the lungs saturating clothes; likewise, in cases of eruptive fevers, measles, scarlatina, r  theln, typhus, and the like, the general exhalations of the sick, and especially so of the convalescing, probably in connection with the desquamation of the skin. The caution which is necessary with regard to such matters must of course extend to whatever may be imbued with them; so that bedding, clothing, towels, and other articles which have been in use by the sick, do not become sources of mischief, either in the house to which they belong, or in houses to which they are conveyed. Moreover, in typhoid fever and cholera, the evacuations should be regarded as capable of communicating a similarly specific and infectious property to any night-soil with which they may be mingled in privies, drains, or cess-pools (THIERSCH). This danger of multiplying the sources of communicating disease must be guarded against by the chemical destruction, decomposition, or disinfection of all the intestinal evacuations as soon as they are passed from the bowels, and certainly before they are thrown away, and so "let loose upon the world." Above all, they must never be cast where they can run or soak into sources of drinking-water.

12. All reasonable care should be taken not to disseminate disease by the unnecessary association of persons suffering from the specific communicable diseases, either with healthy persons, or in wards of hospitals where patients suffering with other diseases are being treated. This care is requisite, not only with regard to the sick-house, ward, hospital, or ship, but likewise with regard to day schools, places of public resort, courts of justice, and other places where members of many different households are accustomed to meet.

13. Where dangerous conditions of residence cannot be promptly remedied, it will be best that the inmates, while unattacked by disease, remove to some safer lodging. If disease begins in houses where the sick person cannot be rightly circumstanced and tended, medical advice ought to decide on the propriety or fitness of removing him to an infirmary or hospital. In extreme cases, special infirmaries may become necessary for the sick, or special houses of refuge for the endangered.

14. The questions of quarantine ought to be decided by the

circumstances of the special case, the preceding principles being kept in view.

15. Privation, as predisposing to disease, may require special measures of relief.

16. In certain cases special medical arrangements are necessary. For instance, as cholera in this country almost always begins somewhat gradually, in the comparatively tractable form of what is called "premonitory diarrhoea," it is essential that, where cholera is epidemic, arrangements should be made for affording medical relief without delay to persons attacked even slightly with looseness of the bowels. So again, where small-pox is the prevailing disease, it is essential that all unvaccinated persons (unless they previously have had small-pox) should very promptly be vaccinated; and re-vaccination should also be offered, both to persons above puberty who have not been vaccinated since childhood, and to younger persons whose marks of vaccination are unsatisfactory.

17. It is always to be desired that the people should, as far as possible, know what real precautions they can take against the disease which threatens them; what vigilance is needful with regard to its early symptoms; and what, if any, special arrangements have been made for giving medical assistance within the district. Especially in the case of small-pox or of cholera, such information ought to be spread abroad by means of printed bills or placards. In any case where danger is great, house to house visitation, or personal inspection of all by discreet and competent persons, may be of the utmost service, both in quieting unreasonable alarm, and in leading or assisting the less educated and the destitute parts of the population to do what is needful for safety.

18. These memoranda relate to occasions of emergency. The measures suggested must be regarded as of an extemporaneous kind. Permanent provisions for securing Public Health have not been in express terms insisted on. In proportion as a district or number of individuals, such as an army or a regiment, are habitually well cared for by its sanitary authorities, the more formidable emergencies of epidemic disease are not likely to arise.

As addenda to these memoranda, the following rules, the observance of which is enjoined by the government of the

London Fever Hospital, might well be adopted under similar circumstances in military and in civil hospitals:—

1. It is of the utmost importance to the sick and their attendants that there be a constant admission of fresh air into the room, and especially about the patient's bed, care being taken to prevent the wind from blowing directly on the patient.

2. Attention to cleanliness is indispensable. The linen of the patient should be often changed, and the dirty clothes, &c., immediately put into fresh cold water, and afterwards well washed. The floor of the room must be cleansed every day with a mop, and all discharges from the patient immediately removed, and the utensils washed.

3. Nurses and attendants ought to endeavour to avoid the patient's breath and the vapour from the discharges.

4. Visitors must not go near to the sick, nor remain with them longer than is absolutely necessary: they should not swallow their spittle, but clean the mouth and nostrils when they leave the room.

5. No dependence must be placed on vinegar, camphor, or other supposed preventives, which, without attention to cleanliness and admission of fresh air, are not only useless, but by their strong smell render it impossible to perceive when the room is filled with bad air or noxious vapours.

Processes of Disinfection.—These processes have been recommended by Professor Miller, of King's College, London. They cannot supply the place of cleanliness, ventilation, and drainage. They are artificial, and are used for exceptional purposes, the great natural disinfectant being *fresh air*, abundantly and uninterruptedly supplied.

1. For purposes of artificial disinfection, the agents which most commonly prove useful are, chloride of lime, quicklime, Condyl's manganic compounds, and carbolic acid. Metallic salts, especially perchloride of iron, sulphate of iron, and chloride of zinc, are under some circumstances applicable. In certain cases chlorine gas or sulphurous acid gas may advantageously be used; and in certain other cases powdered charcoal or fresh earth.

2. If perchloride of iron or chloride of zinc be used, the common concentrated solution may be diluted with eight or ten times its bulk of water. Sulphate of iron or chloride of lime may be used in the preparation of a pound to a gallon of water, taking care that the water completely dissolves the sulphate of iron or

has the chloride of lime thoroughly mixed with it. Condyl's stronger fluid (red) may be diluted with fifty times its bulk of water; his weaker fluid (green) with thirty times its bulk of water. Where the matters requiring to be disinfected are matters having an offensive smell, the disinfectant should be used till the smell has entirely ceased.

3. In the *ordinary emptying of privies or cess-pools*, use may be made of perchloride of iron, of chloride of zinc, or of sulphate of iron. But where disease is present, it is best to use chloride of lime or Condyl's fluid. Where it is desirable to disinfect before throwing away the evacuations from the bowels of persons suffering from certain diseases, the disinfectant should be put into the night-stool or bed-pan when about to be used by the patient.

4. *Heaps of manure* or of other *filth*, if it be impossible or inexpedient to remove them, should be covered to the depth of two or three inches with a layer of freshly burnt *vegetable* charcoal in powder. Freshly burnt lime may be used in the same way, but is less effectual than charcoal. If neither charcoal nor lime be at hand, the filth should be covered with a layer, some inches thick, of clean dry earth.

5. *Earth near dwellings*, if it has become offensive or foul by the soakage of decaying animal or vegetable matter, should be treated on the same plan.

6. *Drains and Ditches* are best treated with chloride of lime, or with Condyl's fluid, or with perchloride of iron. A pound of good chloride of lime will generally well suffice to disinfect 1,000 gallons of running sewage; but of course the quantity of disinfectant required will depend upon the amount of filth in the fluid to be disinfected.

7. *Linen and washing apparel*, requiring to be disinfected, should, without delay, be set to soak in water containing, *per gallon*, about an ounce either of chloride of lime or of Condyl's red fluid: the latter, as not being corrosive, is preferable. Or the articles in question may be plunged at once into boiling water, and afterwards, when at wash, be actually boiled in the washing water.

8. *Woollens, bedding, or clothing*, which cannot be washed, may be disinfected by exposure for two or more hours in chambers constructed for the purpose, and heated to a temperature of 210° to 250° Fahr.

9. For the disinfection of *interiors of houses*, the ceilings and walls should be whitewashed with quicklime. The wood-work should be well cleansed with soap and water, and subsequently washed with a solution of chloride of lime, about two ounces to the gallon.

10. *A room no longer occupied* may be disinfected by sulphurous acid gas, or chlorine gas,—the first by burning in the room an ounce or two of flowers of sulphur in a pipkin; the second by setting in the room a dish containing a quarter of a pound of finely powdered black oxide of manganese, over which is poured half a pint of muriatic acid, previously mixed with a quarter of a pint of water. In either case the doors, chimney, and windows of the room must be kept carefully closed during the process, which lasts for several hours.

CHAPTER V.

DETAILED DESCRIPTION OF THE MIASMATIC ORDER OF ZYMOTIC DISEASES.

SECTION I.—ERUPTIVE FEVERS.—*Exanthemata*.

SMALL-POX.

LATIN, *Variola*; FRENCH, *Variole*; GERMAN, *Blattern*—Syn., *Menschenpocken*; ITALIAN, *Vajuolo*.

Definition.—*Small-pox in man is the product of a specific and palpable morbid poison, which is reproduced and multiplied during the course of the malady. After a definite period of incubation a remittent fever is established, and followed by an eruption on the skin, and sometimes on the mucous surfaces, with other concomitant and occasionally succeeding affections. The eruption on the skin passes through the stages of pimple, vesicle, pustule, scab, and leaves marks or cicatrices on its site. The disease runs a definite course, and, as a rule, exhausts the susceptibility of the constitution to another attack.*

Pathology.—The theory regarding the development of small-pox is, that a specific poison is absorbed and infects the blood, and after a given period of latency gives rise to “primary fever,” which lasts from two to four days, till the eruption appears,

when the fever for the most part *remits*. The secondary or specific action of the poison of small-pox makes itself obvious by an eruption on the skin, and also sometimes on the mucous membrane of the eyes, nose, mouth, fauces, and great intestine. The eruption runs a given course—namely, *pimple*, *vesicle*, and *pustule*—and when fully out, or at its height, the febrile phenomena, which had remitted, return, and give rise to what is termed the *secondary fever*. The occasionally succeeding morbid conditions are inflammation of the various tissues of the lungs, of the urinary organs, and, lastly, of the areolar tissue of the body generally, which often becomes the seat of an endless number of abscesses.

The occurrence of fever preceding the secondary or specific actions of the poison, or the appearance of the eruption, has scarcely an exception, and, indeed, in some instances it has been of so severe a character as to have destroyed the patient on the first onset. The remission or subsidence of the fever is constant in mild cases, but in the severer forms of the confluent small-pox it sometimes runs on, and is constant. The recurrence of the “secondary fever,” and the exacerbation of the fever in severe cases at the time of the maturation of the pock, is also constant. The cause of this secondary attack has long been a difficulty in the pathology of small-pox. Some attribute the fever to the specific nature of the disease, while others consider it to result from the maturation of the pustules, and to be a *suppurative fever*—*symptomatic*, and dependent upon the local affection.

Another constant phenomenon in the development of small-pox is, that the secondary actions of the poison occasion a peculiar eruption. There are a few rare exceptions, which constitute a variety of small-pox sometimes noticed as the “*variola sine eruptione*.” The affection of the mucous membranes is often wanting in mild cases, though rarely absent in severe ones. The poison is also apt to set up many tertiary actions, as inflammation of the lungs, of the urinary organs, of the eye, and of the areolar tissue. Generally it may be mentioned that the state and appearance of the eruption depends in a great measure upon the type and character of the fever, while the type and character of the fever may be modified by the organic functions and condition of the blood, especially as induced by vaccination.

Pimple 1-2 days
Vesicle 4
Pustule 3
Scab 3

The development of small-pox is traceable through certain stages, namely,—1st. The stage or period of incubation; 2d. The febrile stage, or period of primary fever; 3d. The exudative stage, or period during which the eruption appears and becomes fully developed; 4th. The suppurative stage, or period of secondary fever.

As the eruption, or formation of the small-pox pustule, is undoubtedly a marked characteristic of the disease, it requires particular description. It has itself certain definite stages in its development. It runs a given course of about eleven days, and in its progress undergoes many mutations. It is at first a pimple, then a vesicle, then a pustule, and lastly it forms the scab or crust. These various changes form so many *stadia* of unequal duration. The first, or stage of pimple, lasts from twenty-four to forty-eight hours; the second, or vesicular stage, four days; the pustular stage, three days; while the last stage, or that of scabbing, lasts three days more, making the whole duration of the *normal* pustule ten or eleven days. There are varieties, however, of this disease, in which the formation of the pustule is irregular, as in the *confluent* and *horn* small-pox. In the latter the two last stages are singularly shortened, or absent altogether.

When the eruption in small-pox is of the "*distinct variety*" (*Syn.*, "discrete"), its first appearance consists of a number of small red pimples, about the size of a pin's head, more or less numerous, but separate and distinct from one another, and scarcely salient. On the second or third day of the eruption the second stage towards the development of pustules commences; and a small vesicle, which gradually enlarges, bound down and depressed in the centre, or umbilicated, forms on the apex of each pimple, and contains a clear whey-coloured fluid. This vesicular stage lasts about four days, when the vesicle matures or "ripens" into a pustule.

This process is so gradual that, if you examine the pustule closely about the fifth or sixth day, you may see, at least in many, two colours, viz., a central whitish disc of lymph, set in, or surrounded by, a circle of yellowish puriform matter. In truth, there is in the centre *a vesicle*, which is distinct from the pus, so that you may puncture the vesicular portion, and empty its contents, without letting out any of the pus; or you may puncture the part containing the pus, and let *that* out without evacuating the contents of the vesicle. The vesicles have even, by careful dis-

section, been taken out entire" (WATSON). The adherence of the altered cuticle to the cutis at some points, and its separation at others, produces the little compartments or dissepiments spoken of by some writers. These cavities are usually irregular in shape; and all who have examined these multilocular cavities agree in describing the existence of a white substance in them, of the consistence of pulp or thick mucus, and which at first was supposed to be the specific exudation of small-pox. It is now ascertained that it is no pseudo-membrane, but is composed of the deeper and softened layers of the epidermis. This "disc" of softened epidermis covers the interior of the pustule, and extends from the centre to the raised circumference of the pustule in diverging rays, forming part of six or eight chambers of nearly equal size. In the structure of this disc the following elements are distinguishable from without inwards,—(1.) Large flat cells; (2.) Large cells not so flat, but more globular, with nuclei; (3.) Nearest the cutis are the cells and tissue of the *rete mucosum* (GRUBY, GLUGE, RAYER GUSTAV. SIMON, besides other observers of more early date). Will not some delicate process of organic analysis tell us what the *active principle* of the specific virus of small-pox is,—if it be capable of being so determined? While the maturation of the vesicle into a pustule is going on, a damask red areola forms around each pustule; and as the vesicle fills, the whole face swells, and often to so great a degree that the eyelids are closed. While the maturation is complete, the "*bride*," which bound down the centre of the vesicle, ruptures, and the pustule now becomes *spheroidal* or *acuminated*. About the eighth day of the eruption a dark spot is seen on the top of each pustule. At that spot the cuticle ruptures, allowing matter to exude, which concretes into a scab or crust; and during this process the pustule shrivels and dries up. The crust is detached between the eleventh and fourteenth days, leaving the cutis beneath of a dark reddish-brown hue—a discoloration which lasts many days, or even weeks. On the face, however, the pustule often penetrates or burrows, so as to cause ulceration of the *rete mucosum*, leaving a permanent cicatrix in the form of a depression or "pit." The cicatrix thus formed, though at first of a reddish-brown, ultimately becomes of a dead white colour.

The small-pox eruption does not appear over the whole body at once, but appears in three successive crops. The first crop

covers the face, neck, and upper extremities, the second the trunk, while the third appears on the lower extremities. There is usually an interval of several hours between each crop; and the later the pustules are in appearing on the trunk and lower extremities than on the face and neck, by so much the later they are in maturing, and in disappearing from those parts. When the eruption on the face is declining, that upon the extremities has scarcely yet arrived at its height, so that the hands and feet are then considerably swollen. This is to be regarded as a favourable sign, in so far as it indicates a certain vigour of constitution.

The number of pustules is very various, sometimes not exceeding five or six over the whole body, more commonly from one to three hundred, and occasionally amounting to several thousands. It has been calculated, if ten thousand pustules be counted on the body, that two thousand at least will be found on the face; and accordingly, the number of pustules on the face being in proportion, those on the other parts of the body furnish a fair estimate of the extent of the disease, and of the danger of the patient.

The pustule is subject to many irregularities, both as to its form and course; which give rise to two very marked varieties of the disease, namely, the *confluent* and the *horn* small-pox. The *confluent small-pox* differs from the *distinct small-pox* in the pimples being small, less prominent, and so numerous that even on the first appearance of the eruption there is hardly any distinct separation between them. The vesicles which form on their appices appear earlier, and their diameters increase more irregularly than in the distinct forms, and often they run one into the other. The pustules, likewise, which are confluent, either remain flat, and do not rise, or, the areolar tissue rupturing, they form large bullæ or bladders in clusters like a bunch of grapes—a rare variety of the disease (*variola corymbosa*)—and are not encircled with the usual red areola round their base; neither do their fluid contents always acquire the yellow colour and thick purulent consistency of the milder disease. Their crusts, moreover, are soft, and do not fall off till many days after the usual period, or not till the eighteenth or twentieth day, or even later. When the desiccation is completed and the crust detached, a deep scar or pit, sometimes an extensive seam, remains, and shows the loss of substance that has taken place, and how destructive has been the process beneath these crusts.

The *horn small-pox* is a variety of the pustule, and is by much the mildest form of the disease. The pustule in this variety passes through the stages of pimple and of vesicle, but on the fifth or sixth day of the eruption, instead of maturing, the pustule shrivels, desiccates, and crusts, and the disease terminates three or more days earlier than in the usual course, and without the occurrence of any secondary fever. This is the form of the disease which so usually follows after vaccination. #

Many other varieties have been described by the older authors, which are seldom if ever now seen—for instance, *black small-pox* (SYDENHAM); a *blood small-pox* (MEAD), or *Hæmorrhagic*, in which “*blood is effused into the vesicles or pustules*,” a *siliquous small-pox* (FRIEND), in which the pustule resembles a small hollow bladder, but contains no fluid. There is one variety, however, which is not uncommon, called the *crystalline* or *pearl pock* (*variolaë crystallinæ*), in which the vesicle continues transparent, seldom matures, and has a tendency to become confluent. Every variety of the eruption, when the disease is severe, may be intermixed with petechiæ. Such are the chief features of the disease, so far as the development of the eruption is concerned.

Varieties and Symptoms of Small-Pox.—The species of small-pox to be described are,—(1.) *The Natural* or *Unmodified Small-Pox*; (2.) *The Inoculated Small-Pox*; (3.) *The Modified Small-Pox*, or *Small-Pox after Vaccination*, or *Varioloid*. Of these in their order; and,

1. *Of the Natural Small-Pox.*

There are several varieties of this species, namely,—1st. The small-pox without eruption (*variolaë sine eruptione*); 2d. The distinct small-pox (*variolaë discretæ*); and 3d. The confluent small-pox (*variolaë confluentes*); 4th. *Semi-confluent*; 5th. *Abortive*; 6th. *Petechial*; 7th. *Hæmorrhagic*; 8th. *Corymbose*.

Symptoms of the Small-Pox without Eruption.—Sydenham and Frank observed in every variolous epidemic that some few persons who have not previously had the small-pox, or, according to Frank, have neither had the small-pox nor been vaccinated, are seized during the time the small-pox is raging, with all the symptoms of primary variolous fever, which having subsided, they have afterwards been found insusceptible of the disease. Sydenham states that he has seen fatal cases of this kind

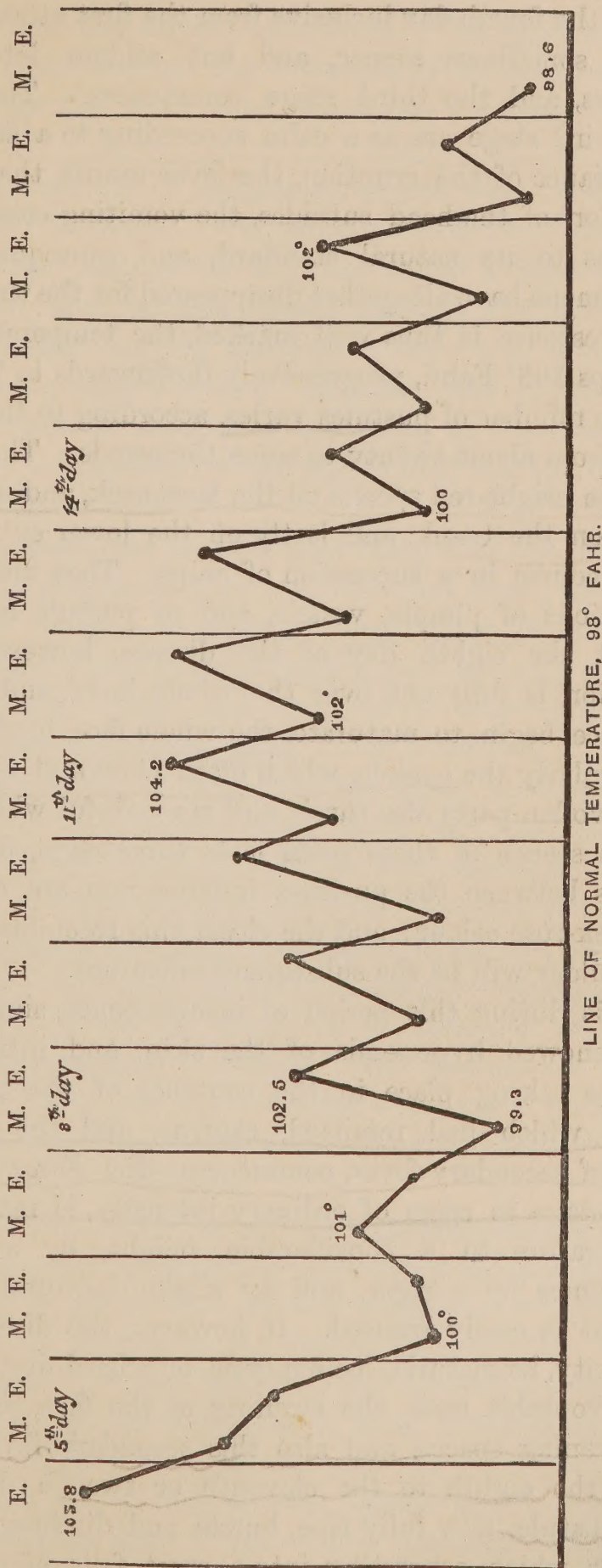
Incubation 10-16 days
2nd stage 4 days

attended with purple spots and bloody urine,—and hence the “*variola sine eruptione*” of authors,—which, when it occurs in the present day, is more usually regarded as a modification of small-pox, probably depending on the influence of vaccination.

Symptoms of the Distinct Small-Pox.—The symptoms of *variola discreta*, or of distinct small-pox, may be divided into *four stages*. The *first stage* comprises the period of incubation or of latency—a period of time which varies according as the poison has been introduced by the mucous or cutaneous tissues. In the former case, or in natural small-pox, for example, the more usual time of latency is from ten to sixteen days; while in the inoculated small-pox the period of latency is from seven to nine days, the extremes, taking both forms of the disease, being from five to twenty-three days. Bärensprung, of Berlin, has lately recorded a most interesting fact, which demonstrates, in a more striking and definite manner, the period of latency; and which appears to be similar in persons who have been vaccinated and in those who have not. He observed seven cases of small-pox, *all* of which were infected from the same source on the same day. In *all* of them the outbreak occurred between the thirteenth and the fourteenth day. Some of them were vaccinated and some were not (*Annalen des Charite Kranken*, vol. xix., p. 103). The *second stage* comprises the primary fever, which commences with the disease, and terminates with the appearance of the eruption. The *third stage* commences with the eruption, and terminates with the appearance of the secondary fever. The *fourth stage* commences with the secondary fever, and includes all the subsequent phenomena.

In the adult the symptoms of the second stage are mainly to be distinguished from those of the first stage of typhus, or other febrile affections, by the characteristic ranges of temperature. There is, however, a great tendency to vomiting, and to pain in the back, and the brain is oppressed, as indicated by drowsiness, stupor, or coma, followed occasionally by convulsions. The ordinary duration of this fever is four days, and it may be sudden in its attack, or be preceded by some days' illness, in which case the most prominent and characteristic symptoms in the adult are severe muscular pains simulating rheumatism, especially in the small of the back, and the frequent occurrence of obstinate vomiting, foreboding a severe form of the disease.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF NATURAL SMALL-POX, COMMENCING WITH THE *THIRD* *STAGE*; THAT IS, FROM THE PERIOD OF THE ERUPTION, ON THE EVENING OF THE *FOURTH* DAY FROM THE BEGINNING OF THE SICKNESS. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).



On the fourth day inclusive from the first attack of the primary fever, sometimes sooner, and but seldom later, the eruption appears, and the third stage commences. The phenomena of the third stage are as a calm succeeding to a storm; for, on the appearance of the eruption, the fever remits, the heat abates, the affection of the head subsides, the vomiting ceases, and the pulse returns to its natural standard, and consequently the febrile phenomena have altogether disappeared for the time. A temporary defervescence is thus well marked, the temperature falling, from perhaps 106° Fahr., progressively downwards to 100° Fahr.

The number of pustules varies, according to the severity of the case, from about twenty to some thousands. They appear first in minute bright-red specks on the face, neck, and upper extremities, then on the trunk, and lastly on the lower extremities, and run their course in a succession of crops. They undergo the various mutations of pimple, vesicle, and of pustule already described. About the eighth day of the disease, however, or when the eruption is fully out over the whole body, and the pustules on the face begin to mature, the whole face, head, and neck swell, particularly the eyelids, which often close and blind the patient; the swollen parts also throb, and are painful when touched. The intumescence of these parts lasts three days, during which the spaces between the pustules inflame, and are of a deep red or damask-rose colour; and the closer this resemblance is seen to be, the milder will be the subsequent affections.

It is during this period of intumescence, simultaneously with the renewed hyperæmia of the skin, and introductory to the change taking place in the contents of the pustule, that the fever, which had remitted, returns, and the *fourth stage*, or that of secondary fever, commences—the *Fever of Suppuration*. This stage, in cases of ordinary intensity, is marked by a rise of temperature to a considerable height, by a frequent pulse, sometimes by a rigor, and by slight delirium, from which the patient is easily aroused. If, however, the disease be of greater intensity, hæmaturia, hæmoptysis, or a hard dry cough, are added. In favourable cases the swelling of the face, the redness of the intervening spaces, and also this secondary fever, having lasted from the eighth to the eleventh or twelfth day, subside, and the pustule, now fully ripe, bursts and discharges a thin yellow matter, which, concreting into a crust, falls off on the fourteenth

or fifteenth day, and the disease terminates. During this somewhat protracted defervescence the temperature sinks gradually, to rise, perhaps, for the third time, when the desiccation takes place.

In the very mild variety of distinct small-pox which was wont to be named the "*horn-pox*," the primary fever is little more than a febricula; the pustules do not exceed half a dozen to two or three hundred; and, having passed through the stages of pimple and of vesicle, they, on the eighth day—*i. e.*, about the usual time of maturation—shrive, desiccate, and crust. The secondary fever, often so fatal, does not recur, so that the convalescence usually commences on the eighth day, and the disease is terminated on the eleventh.

It was once supposed that in such cases the pus of the pustules was absorbed, but it appears that pus does not form, the fluid always remaining serous. In cases of any degree of severity, even in the *distinct small-pox*, the poison acts not only on the skin, but also on the buccal and conjunctival membranes, and produces an exudation on those parts. This additional affection, however, does not appear to aggravate the fever, or to occasion other inconvenience than what arises from the local disease. The buccal eruption is usually preceded and accompanied by soreness of the throat and difficulty of swallowing, and sometimes salivation; but these symptoms do not exceed those of a common sore throat. The exudation upon the mucous membrane is generally resolved without the formation of ulcers, or anything that can be considered a scab or cicatrix. The exudations which form within the eyelids are not attended with much pain, and it is only when the swelling has subsided that the mischief which sometimes takes place is discovered.

A peculiar faint and sickly odour, of a "greasy, disagreeable" kind, and quite *sui generis*, emanates from the small-pox patient during the period of maturation of the pustules. So much is this the case, that Dr. Watson says "one might name the disease at once by the smell." When, however, the disease assumes an unfavourable character, and threatens a fatal termination, the face, which ought to have been intumescent on the eighth day, remains without increase of size, and the spaces which ought to have inflamed are pale and white. The pustules also, says Sydenham, look red, and continue elevated (even after death),

and the saliva, which flowed freely up to this day, suddenly ceases. At this critical period the secondary fever, instead of its usual sthenic character, may assume one of two forms,—namely, either a form like the second stage of typhus, with brown tongue, frequent pulse, and delirium; or the patient may be overwhelmed with the depressing influence of the poison, and sink almost without experiencing a re-action, the pulse being hardly increased in frequency, the heat of the body natural, and the intellect unimpaired. But the patient suffers from an indescribable restlessness, an inexplicable anxiety, some cough, with sickness, a frequent desire to pass urine, and with these symptoms he dies.

Symptoms of the Confluent Small-Pox.—The confluent small-pox is described by Sydenham as beginning with symptoms similar to those of the distinct small-pox, but more violent; the second stage, or primary fever, being attended with more sickness and vomiting, with a higher temperature, with rigors, with more severe muscular pain, with more considerable delirium, and in children often, on the evening before the eruption, by convulsions. This fever is not only more intense than in the distinct kind, but is of shorter duration, and more tumultuous—the eruption appearing more generally on the third day, or even earlier; and the sooner the pustules appear, so much the more confluent is the disease that follows likely to be. The eruption is often preceded by an extensive erythematous or erysipelatous inflammation, and the pimples come out irregularly, or in small clusters, like measles, and are less eminent than in distinct small-pox. "The pustules run together over the greater part of the body."

When the third or eruptive stage is formed, the primary fever remits, but not so completely as in the distinct kind, for the pulse often continues frequent (110 to 120 in a minute), and the temperature does not fall so distinctly, the tongue is white, and even the delirium may recur in the evening. This eruption also has some remarkable characters; for the pustules, especially those of the face, do not rise; they are more irregular and flatter in their forms; and, from their greater number and contiguity, run into each other, or are confluent, sometimes forming bullæ as large as a hen's egg, and sometimes scarcely a portion of healthy skin is visible.

Other symptoms, sometimes seen in the distinct small-pox,

never fail to accompany the second stage of confluent small-pox—namely, sore throat and salivation. The tonsils and the fauces become tumid and red, the face begins to swell, and then the salivary discharge begins either with the eruption or within a day or two afterwards. The discharge of saliva is at first thin and copious, resembling the ptyalism of mercury. About the eighth day, however, it becomes viscid, and is expectorated with difficulty; while in bad cases it either ceases for a day or two, and then returns, or it disappears altogether abruptly; and if the swelling of the face also subsides suddenly, the danger is great. Children are not so liable to this salivation as adults. In them, however, a vicarious diarrhoea often appears, but not constantly; neither does it occur so early in the disease. It is frequently profuse, and often proceeds till the disease terminates. Not unfrequently the larynx and trachea are implicated, even to the larger divisions of the bronchia. There is cough, with hoarseness, painful expectoration, and sometimes complete extinction of the voice. These are most dangerous symptoms.

Watson

It has been stated that, on the appearance of the eruption and the commencement of the third stage, although the fever is mitigated, it does not altogether subside, defervescence is incomplete, and the affection of the head, the frequency of the pulse, and greater heat of the surface, often continue. With these ominous symptoms still present, on the eighth day of the eruption, or the eleventh day of the fever, the fourth stage, or secondary fever, commences, bringing with it new sources of anxiety to the physician and of danger to the patient. Gregory and Watson both consider the eighth day of the eruption as the most perilous day of the disease. Blood often appears in the urine in slight and sometimes in large amount. Renal cylinders are not uncommon. The bladder is affected in a great number of cases, and there is increased mucus. If the urine be retained in torpid and semi-comatose cases, it becomes soon ammoniacal, as in all cases with catarrhal cystitis (Parkes *On the Urine*, p. 262).

D.M.

"The confluent small-pox," says Sydenham, "does not in the least endanger life in the first days of the illness, unless there happens a flux of blood from the urinary passages, or from the lungs. Yet, on the decline of the disease, or on the eleventh, fourteenth, seventeenth, or twenty-first days, the patient is often

brought to such a state that whether he will live or die is equally uncertain. He is first endangered on the eleventh day by a high fever (and the highness of the temperature may indicate the danger), attended with great restlessness, and other symptoms which ordinarily prove destructive, unless prevented by medicine. But should the patient outlive this day, the fourteenth and seventeenth are to be apprehended, for a very vehement fit of restlessness comes on every day towards evening, and there is the greatest difficulty in saving him." The disease is apt to prove fatal by way of apnœa, after the eighth day; but after that period the characters of asthenia supervene.

The fatal symptoms of the fourth stage are, the absence of the usual redness in the intermediate spaces, the non-intumescence of the face, the suppressed salivation, cough, with hæmoptysis, or hæmaturia, and great restlessness. Sometimes other symptoms are added to these, as a brown tongue, delirium, petechiæ, or a black spot in the centre of each pock, scarcely so big as a pin's head; or a disposition to gangrene in the large vesicles. When these symptoms are present, few patients survive the crisis. In some cases, however, the event is favourable, and the patient is restored; but the struggle is sharp, and the convalescence long. In its progress an endless series of abscesses may form, or inflammation of a joint may take place, and produce lameness; ulceration of the cornea, blindness, otitis, or deafness may also ensue; while the deeply-scarred face is a lasting record of the severity of the disease, and of the great danger the patient has survived.

2. *Of the Inoculated Small-Pox.*

Symptoms.—The phenomena which result from the introduction of the variolous poison by means of the cutis differ in many respects from those that occur in the natural small-pox; and they are as follows:—On the day after the operation is performed, little alteration is discovered in the punctured part. On the second day, however, if the part be viewed with a lens, and the operation has succeeded, there generally appears an orange-coloured stain around the incision, while on the fourth or fifth day the part is hard, slightly inflamed, and itches, and a vesicle containing serum is formed on it. About the sixth day some pains and stiffness are felt in the axilla, symptoms which foretell the near approach of the fever and the favourable progress of the

disease. On the seventh day the vesicle becomes more developed and the red areola forms round its base.

The operation having now been performed seven, eight, or nine days (the usual period of latency of the poison when so inoculated), and the vesicle having existed four days, the ordinary symptoms of primary fever appear. This fever lasts three or four days, when the general eruption follows, now called the secondary eruption, the pustules coming out, as usual, in three successive crops, on the face, trunk, and lower extremities. On the day of the general eruption the primary pustule, says Dr. Gregory, is distended with matter, and proceeds on its course, so that it has scabbed when the secondary eruption is only about to mature.

The most remarkable phenomena, however, of the inoculated small-pox are the singular mildness of the fever and the diminished number of the pustules of the secondary eruption. The mildness of the fever is thus instanced by the late Dr. Watson, of the London Foundling Hospital:—"Of the seventy-four persons whose histories I have related, though inoculated with variolous matter in different states, although prepared in so different a manner, and a great number not otherwise prepared than by an abstinence from animal food, not one of them were disordered enough during the whole progress to occasion the least anxiety for the event; not one of them had their eyes closed a single day, from the pustules being upon the eyes or near them; none continued in bed an hour longer than they would have done in their best health.

The number of pustules is subject to great varieties, but, with very few exceptions, is much less than in the natural small-pox. In some cases not more than two or three appear; occasionally only the primary pustule is seen; but more generally the number varies from ten to two hundred, the mean being thirty or forty. Such is the general course of the inoculated small-pox. In a few instances, however, the disease that follows this operation is extremely severe, and in a still smaller number it is confluent; and in either case the patient is perhaps destroyed. Many theories have been propounded to explain the singular mildness of the inoculated small-pox, but none of them are satisfactory.

Complications of Small-Pox and Special Morbid Tendencies.—Small-pox having been chiefly studied previous to any sound knowledge of morbid anatomy, or of morbid poisons, the occa-

sional subsequent affections of the disease are still but imperfectly known. About the eighth day in the distinct small-pox, and the eleventh day in the confluent small-pox, a secondary fever is established, and at the same time a new series of phenomena may present themselves in a few severe cases,—as affections of the lungs, of the pleuræ, or of both; of the urinary organs, or of the areolar tissue of the body generally. It is during the progress of this *secondary fever* that frequent opportunities occur for its degeneration into a fatal type. In such cases complete defervescence is never established; but lesions become developed whose advent is capable of being appreciated by careful records of morning and evening temperature during the progress of this *fever of suppuration*. These are the *tertiary affections*, the eruptions and the fever being the *secondary effects* of the specific poison.

M The most frequent affection of the lungs is hæmoptysis, but occasionally inflammation of these organs takes place, generally as pleuro-pneumonia. The mucous membrane, for instance, of the trachea is found often covered with a thick semi-purulent, muciform matter, peculiar to small-pox, irregular or honey-combed at its free surface, and which being removed, the subjacent tissue is found diffusely inflamed. The substance of the lungs also is occasionally found inflamed in every degree, even to purulent infiltration. The pleura also, according to Dr. Gregory, is peculiarly disposed to inflammation, which comes on about the eleventh or twelfth day, for the most part very suddenly, and proceeds rapidly to empyema, sometimes destroying the patient in thirty-six hours. The inflammation of the pleura does not merely run into suppuration, but takes every other form to which it is at any time liable.

X The tertiary action of the variolous poison on the urinary and genital organs is seen in the frequent occurrence of hæmaturia, in the occasional formation of abscess of the kidney, in the occurrence of peripheric and parenchymatous orchitis, and in ovaritis; while its action on the uterus is manifest from menorrhagia in the unimpregnated state, and by frequent miscarriage when the patient is parturient. The areolar tissue of the body generally is also acted upon by this poison. In some cases examined a few hours after death the bodies can with difficulty be laid on the table, the skin being detached by the pressure necessary to raise

Hemorrhage } Hemorrhage } all degrees
 Pleurisy } Pleurisy } rapidly to empyema
 Hamaturia - Abscess -
 Testes } Abscess
 Adipose tissue } Abscess

them; and the serous coat of the intestines separates from the mucous and muscular coats with the greatest facility for many feet, and apparently might be entirely peeled off. In some cases the finger can be thrust through the walls of the heart with ease, as if the muscle of that organ had become unnaturally soft and broken down. This affection of the areolar tissue generally is seen in the great tendency to the formation of abscesses on the subsidence of the eruption; for twenty, thirty, and even more small abscesses will sometimes form on a limb or other part of the body, in most formidable succession, and which, on being opened, are found to contain sanious, or, only in a few instances, laudable pus.

Pyogenic Fever.—In a case of septicæmia, occurring during the course of confluent small-pox, examined by Dr. Parkes, the disease ran its course well till the eleventh day, when there was shivering; on the following day there was bilious vomiting; on the fourteenth day there was sudden pain in the right wrist, and swelling of many joints; and on the following day there were all the well-marked symptoms of pyæmia. A daily examination of the urine showed the remarkable fact that the amount of sulphuric acid passed continued progressively to increase daily—rising from 23·8 grains to 44·4 grains (Parkes *On the Urine*, p. 267).

Sequelæ.—The different lesions that have been mentioned are not the only miseries from which the patient may suffer; for these are often followed by sequelæ even more formidable than the preceding phenomena, as blindness, deafness, or lameness. With respect to blindness, it is generally supposed that pustules form on the conjunctiva or cornea, the inflammation then extending to the deeper-seated parts, and thus destroying the eye. Mr. Marson, formerly surgeon to the Small-Pox Hospital, says that, according to his experience, “The eye seems to possess a complete immunity from the small-pox eruption, and that although it sometimes extends to the inner margins of the eyelids, the particular local affection that causes the destruction of the organ of vision in variola begins generally on the eleventh or twelfth day, or later, from the first appearance of the eruption, and when the pustules in every other part of the body are subsiding. It comes on after the secondary fever has commenced, with redness and slight pain in the part affected, and very soon an ulcer is formed, having its

seat almost invariably at the margin of the cornea. This continues to spread with more or less rapidity, and the ulceration passes through the different layers of the cornea, until the aqueous humor escapes, or till the iris protrudes. In the worst cases there is usually hypopion, and when the matter is discharged, the crystalline lens and vitreous humor escape. In some instances the ulceration proceeds very rapidly: I have, more than once, seen the entire cornea swept away within forty-eight hours from the apparent commencement of the ulceration; and, what is singular, now and then the mischief goes on without the least pain to the patient, or his being aware that anything is amiss with his eyes." Further, he calculates that in 1,000 cases 26 had ophthalmia, or about 1 in 39; and of these 11 lost an eye each, or 1 in about 100.

The inflammation of the buccal membrane may extend to the Eustachian tube, causing suppuration of the ear, and sometimes permanent deafness. It may spread also to the glottis; and the patient has been known to die suffocated by effusion into the areolar tissue around it, causing occlusion of the aperture. Sometimes it has terminated in ulceration, with the loss of a portion of the nose, or in caries of the jaw-bone, or in enlargement of the glands of the neck.

The soreness of the fauces and tonsils is often associated with pustules on these parts; and the tongue, the roof of the mouth, the inside of the cheeks, the uvula, and the *velum palati* may be covered with an eruption like pustules; and it has been much disputed whether the eruption forms on any other part of the mucous membrane. As a general principle, it does not: but Martinet found, in a man that died on the eighth day, the rectum covered with what he supposed to be variolous pustules. Rostan has seen the alimentary canal garnished with pustules similar to those of the mouth, from the œsophagus to the rectum. Sir Gilbert Blane also met with pustules on the mucous membrane of the intestines in two persons who died in the West Indies; and Rayer has given a plate representing pustules on the mucous membrane of the trachea. Dr. Mead's experience has made him state that, "I myself have seen subjects in which the lungs, brain, liver, and intestines were thickly beset with pustules." Dr. Pitzholdt, in the *Morbid Anatomy of Small-Pox*, writes that he has seen the peritoneum covering the liver

and the spleen presenting appearances which he felt justified in regarding as the product of small-pox.

The pustules which form on the mucous membrane of the intestine, however, have not been very distinctly studied either as to their course or phenomena. Rayer terms them *rudimentary* pustules; and Dr. Watson believes the statement, that such pustules exist, to be a mistake.

A case of small-pox recorded by Dr. George Patterson, of Edinburgh, was examined by one of the most learned and discriminating pathologists of the day, Professor W. T. Gairdner. He observed pustules on the mucous membrane of the colon, and pronounced them to be identical with the pustules on the skin (*Edinburgh Monthly Journal*, 1849, p. 549). Still it appears to be doubtful whether such eruption on the mucous membrane of the intestine is not the same as that seen in cholera cases, extending (as I have frequently seen it do in cases I examined in the hospitals at Scutari, in 1855) throughout the whole intestinal tract. The appearance of eruption in such cases is due to the solitary mucous glands, which are filled with exudation, not of a purulent kind, but having all the external appearance of pustules.

Such are the pathological phenomena which occasionally complicate small-pox. Death, however, not unfrequently anticipates their action, and destroys the patient during the primary fever, and before any of them are set up.

3. *Of the Small-Pox after Vaccination—Varioloid, or Modified Small-Pox.*

Symptoms, Course, and Modifications.—It has been already noticed that during the epidemic prevalence of small-pox, even before vaccination was known, cases of small-pox occurred in a very modified form: such as the occurrence of variolous fever without the eruption (*variola sine variolis vel eruptione*), or the occurrence of small-pox in which the eruption continued vesicular (*the crystalline pock*), and, lastly, the occurrence of small-pox in which the vesicles dried up instead of becoming mature pustules, and known as stone-pock, horn-pock, wart-pock (*variola verrucosa vel cornea*). Modern pathology now regards these varieties as the result of the modifying influence of vaccination: and they may now be all described and classed under the common name

varioid. Comparative mildness of symptoms and course is their great characteristic, the pustules being "cut short in their development by vaccination or a previous attack of small-pox." There appears to be every variety in the nature of the modification, of which the principal are:—

1. A fever of three days, without eruption, affecting people during variolous epidemics.

2. A high and severe fever, followed by a very mild eruption, sometimes only a single pock: the slight proportion which the amount of eruption bears to the severity of the preceding fever is perhaps the most marked characteristic of varioid.

3. The occasional appearance of a scarlet efflorescence like that of scarlatina or roseola, preceding the appearance of the proper pimples, which occur as a very scanty crop.

4. In some rare instances the eruption is confluent, but does not advance beyond the development of a pimple or vesicle, and begins to dry on the fourth or fifth day of the eruption, forming a small hard tubercle, which soon disappears.

5. Sometimes the eruption is pimple, vesicle, and pustule at one time in the same case.

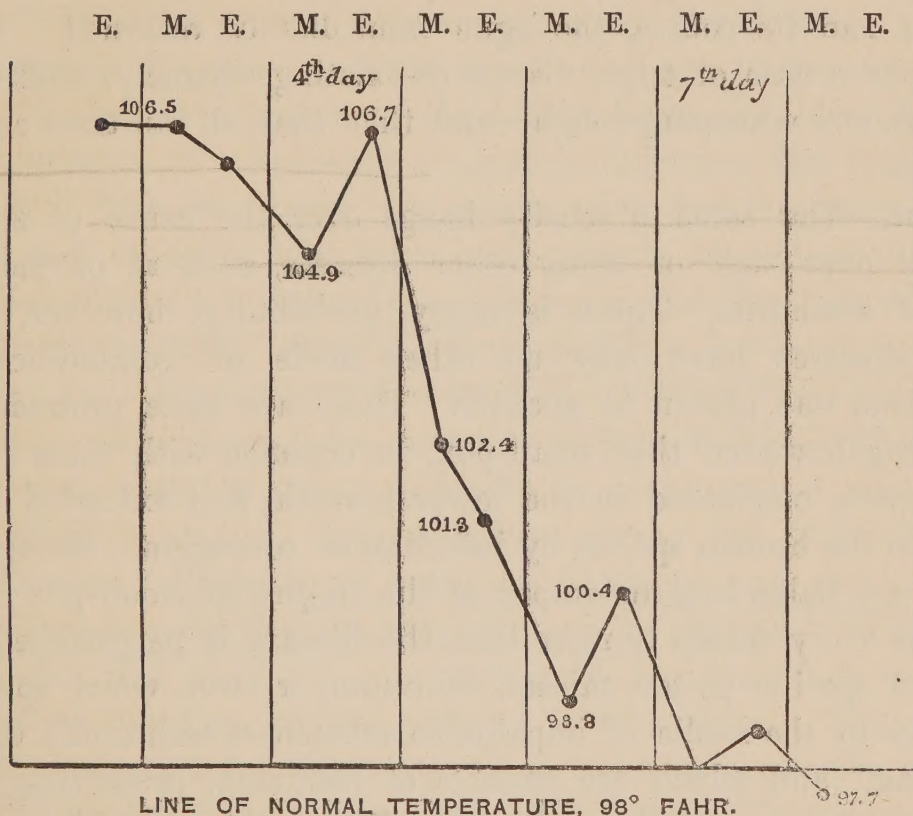
6. Sometimes the eruption runs its regular course, but stops sooner, sometimes on the sixth or seventh day, instead of the eighth or ninth. In general, it may be stated that the severity and fully developed state of the disease is in proportion to the length of time which elapses from vaccination (COPLAND).

7. The varioid eruption wants the peculiar odour of natural small-pox, and secondary fever is very rare.

8. Other eruptive affections—such as measles, scarlatina, purpura—materially modify the course and symptoms of small-pox.

Generally, it may be stated that, after an intense continuous fever, lasting a few days, a final exacerbation terminates the fever suddenly and simultaneously with the development of the small-pox pimples. A rapid and perfect defervescence then ensues, the temperature decreasing seven or even more degrees (Fahr.) within thirty-six hours. From this event the patient remains entirely free from fever—*provided there exists no serious complication*—in spite of the continuous and progressive development of the small-pox pimples into pustules, and even in spite of the successive eruption of new pimples.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF SMALL-POX MODIFIED BY VACCINATION. THE RECORDS INDICATE MORN-ING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE SECOND DAY (Wunderlich).



LINE OF NORMAL TEMPERATURE, 98° FAHR.

Exhaustion of Susceptibility.—The small-pox has the property, in common with measles and scarlet fever, of exhausting, on the first attack, the susceptibility of the constitution to the future actions of the poison. This law, however, is not without some exceptions, and in an epidemic at Marseilles, Bosquet considered that one person in one hundred was attacked a second time with small-pox. In some few instances even a second attack has no protective influence. Dr. Roupel says he met with an instance in which small-pox occurred three times in the same person. The lady of a Mr. Guinnett had it five times. Dr. Matson speaks of a lady who had it seven times; while Dr. Baron mentions a surgeon of the South Gloucestershire Militia who was so susceptible that he took small-pox every time he attended a patient labouring under that disease.

Co-existence of Small-Pox with other Morbid States.—The variolous poison is capable of *co-existing* with many other poisons, and also of influencing their actions, and of being reciprocally influenced

by them. Dessessarz has seen variolæ co-exist with scarlatina and with whooping-cough; Cruickshanks with measles; Frank with psora; and Dimsdale with syphilis. A patient was admitted into St. Thomas's Hospital with tertian ague, writes Dr. Williams; the ague subsided and the small-pox appeared. The small-pox having run its course, the ague immediately returned. Ring mentions a case of triple disease co-existing—namely, small-pox, measles, and whooping-cough—and that they all ran their course together.

Cause.—The same obscurity hangs over the cause of small-pox as over that of many other diseases, such as of measles and of scarlatina. There is every probability, however, that these diseases have now no other mode of communication than from one person to another. There are some grounds for believing, however, that small-pox, in common with some other distempers, originated in the lower animals, and extended from them to the human species by infection or contagion. Sheep, we know, are liable to a distemper of the nature of small-pox; and there is every reason to infer that the disease is perpetuated by its own specific poison, miasm, effluvium, or virus, which spreads it about by the media of impalpable substances technically called "*fomites*," and which are capable of receiving, preserving, and carrying the germs of the disease. By such impalpable means the disease has been propagated since its first appearance in the world. The poisonous material of small-pox is given out from the mucous and cutaneous surfaces of a patient, especially from the lungs and skin, from the exhalations, the secretions, the excretions, the matters in the vesicles and pustules, and the scabs. These all contain the noxious germs of the disease, which may attach themselves to bed-clothes, body-clothes, and especially to woollen, cotton, and felted articles. Such stuffs retain the specific poison for a very long but undetermined period: any number of years, so far as is known—just as the hat, cap, and coat worn in a dissecting-room retain the peculiar effluvia of that place for a very long period.

It is not yet determined at what period this poison is first generated by the patient's person, whether during the primary fever, or not till after the eruption has appeared; but it probably begins to form and multiply during the primary fever. Generally, it may be stated that the poison is most powerful when it is

most manifest to the sense of smell; that the dried crusts of the pustules or scabs possess the power of communicating the disease, and retain this power for a very long time. It is unsafe for a susceptible person—*i. e.*, a person who has not been vaccinated, or has not had small-pox—to be in the same room, or in the same house, with a patient labouring under the disease. It has been caught by passing a child ill of small-pox in the street; so that “to expose a person in the public highway, infected with this contagion, is considered a common nuisance, and indictable as such.” The dead body of a variolated person is equally infectious, and students who have been near it when brought into the dissecting-room have in consequence had the disease communicated to them, although they may not have touched the body (CÆSAR HAWKINS). The *infecting distance*, therefore, must be many yards around the patient’s person: indeed, with every precaution, there is great difficulty in preventing it spreading from ward to ward in large hospitals during the prevalence of the disease. “There is no contagion so strong and sure as that of small-pox: none that operates at so great a distance” (WATSON).

The fact that small-pox is communicable has been fully demonstrated by the once general practice of inoculation. The poison by this operation has been proved to exist in the serum, in the pus, and in the crusts of the small-pox pustule. There is no law more singular and unexpected, in the whole range of morbid poisons, than that the introduction of the variolous poison, by means of the cutaneous tissue, should produce an infinitely milder disease than when the same poison is absorbed by a mucous tissue. Then the poison seems to be much more uncontrollable in its operations, as in the case when it affects a person who breathes an infected atmosphere compared with one who has been *inoculated* with the small-pox poison inserted beneath his cuticle through a puncture of the skin. Several explanations are put forward, namely,—(1.) That the small quantity of the poison conveyed by inoculation into the blood may make the difference; (2.) That the disease is milder when the poison is admitted through the cutaneous than through the mucous tissues; (3.) It may be held that in passing through the absorbent mucous membrane the poison is not only admitted in large quantity, but its potency may be increased and its amount multiplied by the living cells of the mucous membrane through which it passes.

The causes which predispose to small-pox or increase the susceptibility of infection are,—(1.) A very early age. (2.) Not having had the disease before. (3.) Not having been vaccinated: such are called “unprotected persons.” (4.) Peculiarity of constitution—*e. g.*, the Negro and dark races. (5.) Fear of infection. (6.) Epidemic influence.

It is gratifying to know that of recent years the prevalence and mortality of small-pox in this country is greatly less than was wont to be. Dr. Farr tells us that, for the three years previous to 1855, out of every 1,000 deaths from all causes, only 7·607 were from small-pox.

Prognosis and Causes of Death.—The prognosis of the natural small-pox is always most grave. The danger may be measured, to a certain degree, by,—(1.) The quantity and confluence of the eruption; (2.) The state of the circulating fluids; (3.) The presence and nature of the complications, especially those of the respiratory organs and nervous centres; (4.) Age, and habit of body of the patient; (5.) Nature of the epidemic constitution which may prevail.

Natural small-pox in unprotected persons is generally very fatal. The deaths average one in three. The fully formed confluent small-pox is always very dangerous. About one in ten die of distinct natural small-pox; and one to three per cent. only of small-pox after inoculation or after vaccination. The calculation of the proportionate number of deaths, however, appears to have greatly varied in different years.

There are certain signs regarded as unfavourable,—for example: excessive lumbar pains continuing; the persistence of vomiting after the appearance of the eruption; the occurrence of delirium, convulsions, or coma in adults during the primary fever; great confluence and simultaneous appearance of the eruption over the whole body. Such unfavourable signs are not necessarily fatal; but unfavourable signs which appear during secondary fever forebode, with greater probability, a fatal end. These are:—the absence of the usual redness in the intermediate spaces; the distribution of petechiæ in the interstices; the development of a black spot hardly so large as a pin’s head in the centre of each pustule; a livid or purple colour of the pustule; a disposition to gangrene in the larger vesicles; imperfect development of the pustules, or their sudden subsidence, without remission of symp-

W. N.

toms; sudden suppression of salivation; sudden suppression of urine; hæmaturia; cough with hæmoptoë; absence of swelling in the hands and feet when the eruption is copious; tendency to the formation of abscesses (*pyogenic fever*) after desquamation has commenced; congestive pneumonia, or bronchitis, with livid lips, face, or extremities, with hoarseness or complete aphonia. Recovery may take place even although the first-mentioned of these unfavourable signs exists; but convalescence is likely to be retarded by ulcerations of the cornea, asthenic ophthalmia, purulent deposits in the joints, ulceration of cartilages, otitis, abscesses and suppuration in the areolar tissue under the skin.

The development of scrofula and phthisis is apt to follow the disease, even though no unfavourable symptoms occur. In pregnant women the disease is always dangerous, often fatal, and almost always produces abortion; and the foetus so parted with not unfrequently bears evidence of small-pox upon the skin.

The most common causes of death are due to combinations of the unfavourable signs already noticed; and, according to Dr. Gregory's observations at the Small-Pox Hospital in 1828-29, the greatest number die on the eighth day of the eruption; or the eleventh day of the fever is the most fatal period. In private practice, Dr. Wood, of Pennsylvania, considers the period between the twelfth and eighteenth day as the most dangerous to life. The greatest mortality from small-pox is in the early periods of life,—for example, before the fifth year. Dr. Farr estimates that out of every 100 deaths from small-pox, 75 are below that age.

Diagnosis.—It is not possible to distinguish, except by careful records of the temperature, the primary fever of small-pox from that incident to many other diseases with eruptions, or from the first stage of continued fever. It is for the most part characterized by excitement rather than depression; and in the adult the muscular pains and pains in the back and loins are more severe and intense than in ordinary fever. The pain of the back is central in its position—a *spine-ache*—and is less affected by change of posture than the pain which is characteristic of lumbago, which affects the muscles at the side of the spine (often on one side only), and which is much aggravated by movement (BARCLAY). If vomiting occurs, which cannot be ascribed to any obvious cause, and persists till a papular eruption appears on the

third or fourth day, with a remission of the febrile symptoms, little doubt can exist as to the variolous nature of the disease.

The diseases with which it may be, at first, confounded are, *petechial eruptions*, *measles*, and *chicken-pox*, and the *secondary pustular eruptions of syphilis*.

The nature of the fever, the character of the eruption, and the absence of any tendency to suppuration, are sufficient to distinguish *petechiæ* from variolous eruption.

Small-pox is to be distinguished from *measles* by the symptoms, as well as by the form and successive changes of the eruption. Crescentic patches, terminating in desquamation on the fourth day, characterize *measles*, as compared with small-pox, the eruption of which, even although it may be at first in efflorescent patches, never fails to become vesicular and pustular, proceeding to suppuration or blackening on the eighth day—a process which never fails to be attended by secondary fever.

It is more difficult to diagnose between varioloid and varicella, or chicken-pox. The chief difference consists in the eruption of *chicken-pox* presenting a vesicular character, which it retains; and it does not proceed to suppuration, but completes its course in five or six days, with a mild and short symptomatic fever.

The combination of mercury, scrofula, and syphilis often gives rise to cutaneous eruptions attended with fever, which may, in the first instance, be mistaken for small-pox. The eruption, however, is more tedious in its development, irregular in its course, and is persistent. It must, therefore, be distinguished by the history of the case, the long duration of the eruption, and the deep red or copper colour it generally presents.

Treatment.—Since the first accounts by the Arabian physicians of the ravages of small-pox in Mecca, the history of this disease may be arranged in three great eras, each of which is characterized by remarkable epochs, and a fourth may be said to be now becoming apparent. The first of these eras is marked by an improvement in the treatment of small-pox. In few diseases has medical opinion undergone a more obviously beneficial change. To Sydenham is due the merit of this revolution in medical practice. The second era is marked by the discovery of the singular and beneficial phenomenon that the virulence of the poison of *small-pox* is greatly mitigated by introducing or ingrafting the disease into the system, through

the cutaneous tissue, thereby causing the transference of the disease from one person to another, by inoculation. To Lady Mary Wortley Montague is due the merit of having introduced the practice of inoculation into this country in 1722—a deed which must be considered as one of great heroism, when measured by the knowledge possessed by physicians in those days. The third great era is marked by the remarkable discovery which has rendered the name of Jenner immortal,—namely, the modifying and protecting influence of vaccination. He found that a certain disease in the cow, known as the *cow-pox*, could be transferred to the human subject by inoculation; and that, having been so transferred, it modified, to a considerable extent at least, the course of the disease, if it did not altogether prevent the occurrence of small-pox in its natural state in the human subject.

A fourth era may be said to have commenced in this country almost imperceptibly. It may be described as a period of transition, marked in this country by doubt and scepticism as to the efficacy of vaccination, tending to propagate an erroneous popular belief; and consequently, the ineffective adoption of means which practically have been proved to be sanative in the highest degree. In other countries, on the contrary, and especially in Central Europe, this period is marked by implicit faith in the virtues of vaccination, and the successful legal enforcement of this sanative measure.

An account of the treatment of small-pox resolves itself, therefore, into the consideration of two topics, namely,—(1.) The usual therapeutic, curative, or sanative treatment of the disease; (2.) The sanitary treatment—*i. e.*, the means of protecting individuals from the *small-pox*; or of modifying the influence of the malady by inoculation or by vaccination. Of these in their order.

1. *Therapeutic, Curative, or Sanative Treatment of Small-Pox.*

The main object, in the first instance, is to prevent, if possible, MB
a copious eruption; for the severity and danger of the disorder 1 M
may be measured, in some degree, by this. The vulgar belief, that “better out than in,” does not apply in the case of small-pox. The great object is to reserve the strength of the patient; and the attentions of an experienced nurse are demanded. A third indication is to watch for and deal vigorously with intercurrent

*R Calomelanos gr 1
Ext. Col. Co gr 1
Gamboge gr 1
Scammony gr 1 p*

75 Al

inflammatory action, which is apt to be set up. The disease is not under the influence of any specific. It must run its course. But it is the business of the physician to assuage the untoward symptoms which may arise, by all the most approved methods of treatment, in accordance with the science of medicine of the present day.

Dietetic and General Treatment.—In the first instance, the course to be pursued is for the physician to act on the defensive, and simply protect his patient from certain injurious influences to which he may be exposed,—such as heating drinks to force out the eruption, which are apt to be given by ignorant and officious friends. Throughout the whole course of the disease, the diet should be strictly limited to slops, sago, arrow-root, and ripe fruits.

The chamber in which the patient lies should be cool, and freely ventilated. The bed-clothes should be light, the body linen daily changed; and, when the disease is long, the patient's back should be often examined, to prevent sloughing. The scalp likewise should be examined, and, if full of pustules, the hair should be cut off, to prevent its matting. If the disease be diagnosed early, however, it is proper to shave the scalp, because the irritation which attends the suppuration of the pustules is thereby diminished, and cold may be more efficiently applied to the head, if necessary. In the early stage of the primary fever, in severe cases more especially, it is necessary to have the bowels well opened in the first instance, and to keep them regular by saline medicine. A cathartic pill, composed of the following ingredients, will be found to be efficient in most cases, especially if aided by a seidlitz powder, given six or eight hours after the pill:—

Two grains of calomel, one grain of the compound extract of colocynth, one grain of gamboge, and one grain and a half of scammony, made consistent with a little aromatic oil.

The bowels must be daily attended to afterwards, and castor oil, or rhubarb, or magnesia, &c., may sometimes be required. Saline diaphoretics, in the form of James's powder; or the "aqua acetatis ammoniæ," to which a grain or two grains of tartar emetic has been added, so as to have $\frac{1}{8}$ th or $\frac{1}{16}$ th of a grain in every table-spoonful of the mixture, is an efficient and cooling

diaphoretic. Spirit of nitric ether, or the nitrate of potass, may be added if required.

The surface of the body, over the hands, face, and feet, may be sponged several times a day with tepid water, with a view to relieve the intolerable itching; but caution is necessary to prevent exposure to cold. Cold-cream, or a liniment of olive oil, glycerine, and lime-water, smeared from time to time over the itching surface by means of a camel-hair pencil, may be found to afford relief; and chlorine lotions are highly spoken of by Eisenmann. With regard to the occurrence of convulsions in children, it is *not* found that opiates, as recommended by Sydenham and Cullen, are expedient. When the children are robust, or previously in good health, local bleedings, by means of one or two leeches to the temples, are more beneficial. Delirium, violent screaming, intolerance of light or sound, heat of head, all of which indicate a tendency to meningeal congestion, still more clearly warrant the application of leeches.

With regard to the propriety of bleeding (general) in adults, it is now well ascertained that it will neither eradicate the fever nor diminish the amount of the eruption. Bleeding is only warrantable if the pulse be full and strong, combined with evidence of inflammatory congestion in the lungs, liver, or brain.

When delirium, with restlessness, wakefulness, and a frequent pulse, is continuous, an opiate is indicated; and, combined with tartar emetic, is most advantageously given. A draught composed of thirty minims of the solution of muriate of morphia, with half a grain of tartar emetic, will be found beneficial in such conditions, and especially when given at bedtime.

Cooling drinks of lemon-juice, tamarinds, neutral effervescing powders, are always agreeable to the patient, who ought also, for the sake of coolness, to be very lightly clothed. After the eruption has fully appeared, this is all which in ordinary cases requires to be done, and if, towards the tenth or eleventh day, there is much restlessness or sleeplessness, an opiate should be given.

When the febrile symptoms do not abate, as they ought, in the regular course of the disease, cathartics may be daily required to keep the bowels open. The most approved are the saline infusion of senna, or the black draught, the compound powder of jalap, combined with calomel and some aromatic powder, such as ginger.

In this disease the bed-clothes ought frequently to be changed, and abundance of cool fresh air supplied to the apartment. When the state of the skin *alone* seems to keep up the febrile irritation, an antimonial opiate may allay irritation and procure sleep, after which a cathartic may be given with advantage in the morning.

X In the complications which sometimes ensue, such as *inflammation of the throat and base of the tongue*, opiates are found to be injurious. The general treatment must be by cathartics or purgative clysters, if swallowing is difficult. In the other inflammations, however, opiates are of the greatest service, provided the symptoms be not of cerebral oppression; and local blood-letting is always to be preferred to general. In bronchitis, nauseating doses of antimony every hour sometimes procure relief; and if relief does not follow in the course of thirty to thirty-six hours, doses of calomel and opium ought to be given every second hour till three doses have been taken, each consisting of *two grains of calomel and half a grain of opium.* If the symptoms are not then relieved, this remedy need not be carried farther.

(9) In the advanced stage of the secondary fever the strength of the system requires maintenance and support; because the abundant suppuration and extensive cutaneous irritation combine to exhaust the strength, as shown by the weakened pulse, the dark and dry tongue, blueness, paleness, or coldness of the extremities. Tonics, stimulants, and generally nutritious diet, are now called for. Quinine, mineral acids, malt liquors, especially the light bitter ales, wine, and even brandy, may be demanded. The diet should consist of milk, strong animal broths, eggs, raw or lightly boiled, according to the discretion of the physician, regulated by the digestive powers of the patient.

To prevent the face from being seamed, scarred, or "pitted" by the suppuration of the pustules, has taxed the ingenuity of physiologists and physicians. It has been stated that the influence of the atmospheric air is essential to the development of the pustules, and, accordingly, anything which would effectually exclude this influence would prevent the occurrence of a scar. But it is evident that the chance of scars can only be diminished by those means which are calculated to allay the general violence of the disease. When the eruption is severe, it is almost impossible to prevent the formation of "pits," because the depression

results from the expulsion of a small slough; and the more mild the suppurative inflammation can be rendered, so in proportion will the chance of "pitting" be diminished.

The local means adopted to prevent "pitting" may be shortly stated as follows:—

1. To open each individual pustule after suppuration has commenced.
2. To cauterize the pustules with nitrate of silver.
3. To employ both methods—that is, to open each of the pustules when it becomes vesicular, and introduce a strong solution of nitrate of silver into the cavity of the vesicle. At the end of a week scales fall off, and no pit is left. Or, lastly, to paint the face with a solution of nitrate of silver, in the proportion of one drachm of the nitrate to the ounce of water.
4. The application of a mercurial plaster, with the view of producing resolution of the *papulæ*. The preparation in use for this purpose at the Children's Hospital in Paris consists of 25 parts of mercurial ointment; 10 parts of yellow wax; 6 parts of black pitch.
5. Sulphur ointment applied several times a day.
6. Calamine mixed with olive oil, to form a coherent crust (BENNETT).
7. Tincture of iodine, painted over with a brush.
8. Saturated solution of gutta percha in chloroform (DRS. GRAVES and WALLACE).
9. To smear the face over with common olive oil.

All of these applications are for the most part applied to the face, the hands, and the arms only.

The severity and the mortality of small-pox has led many to think of means by which the disease might be completely extirpated. This leads us to consider—

2. *The Prophylactic, Sanitary, or Preventive Treatment of Small-Pox.*

Fifty years ago it was generally taught, among English physicians, that small-pox attacked the same individual only once in the course of life, and that its double occurrence in the same person was either very rare or next to impossible. The observations of Drs. Willan, John Thomson, Mr. Cross, Dr. Barnes, Dr. Craigie, and others since the time of these eminent physicians, lead to the following general conclusions:—

1. Small-pox, though in general attacking the same individual

only once during the course of life, may, however, affect him a second, and even a third time.

2. This happens much more commonly when the first attack has been one of mild distinct small-pox than when it has been severe; and if the first attack has been one of confluent small-pox, it is rare for the same individual to have a second attack.

3. It is established by numerous observations, that an attack of any one of the varieties which have been named spurious small-pox or chicken-pox by no means secures the same individual from an attack of confluent small-pox at a subsequent period.

4. Small-pox produced by inoculation does not necessarily secure the individual against an attack of small-pox induced in the natural way.

5. Every previous attack, however, of small-pox, whether natural or inoculated, exercises some modification on that which succeeds. This modification may be various in degree, from very slight and almost imperceptible to very conspicuous and remarkable. In this modification the symptoms of eruptive fever may be mild and of short duration; and the eruption may consist of vesicles or hard pustules, which disappear without suppuration.

6. The most powerful modifying agent on the course of small-pox is the action of the cow-pox on the constitution, or the disease produced by the application of vaccine lymph to the exposed skin. The specific disease so induced, in a large portion of cases, not only renders the individual less likely to be affected by the variolous effluvia, but, if he is affected, changes very much the characters of the disease which may supervene. Though the fever which precedes the eruption in cases of this class be similar in form and equal in degree to that by which the inoculated small-pox is attended, the eruption is either papuliform or tuberculated, without much surrounding inflammation. A similar eruption is produced when vaccine and variolous matter are inoculated at the same time in the same individual; or when a person who is exposed to the variolous contagion has been inoculated with vaccine lymph early enough to mitigate, but not wholly to supersede, the eruption of small-pox. In such circumstances the vaccine lymph and variolous matter restrain and counteract the operation of each other on the system and on the skin. To these eruptions of modified small-pox the general name of *varioloid* eruptions has been applied.

7. Cow-pox destroys the susceptibility to inoculated small-pox almost entirely; but the susceptibility to the natural disease, or that by inhalation, it does not entirely extinguish. This susceptibility, however, it diminishes in a much greater degree, and much more effectually, than inoculated small-pox does.

8. The susceptibility to second attacks of small-pox,* and attacks of small-pox after vaccination, is principally favoured by the existence of an epidemic constitution of the atmosphere, and by the circumstance of early life, or the age below ten years. If * no epidemic influence exists, the occurrence of second attacks of the disease may not be observed for a long series of years. But if, on the other hand, the atmosphere should possess or acquire an epidemic or variolous constitution, then neither the circumstance of a previous attack of small-pox, nor vaccination, can insure many of those under ten years of age, and not a few between that and thirty, from attacks of small-pox.

The preventive management of small-pox consists,—(1.) In the artificial production of the disease by *inoculation*, or *artificial variolation*; (2.) In the modifying and protective influence of *vaccination*.

Inoculation consists in the application of small-pox matter or virus to the surface of the corium, exposed by a puncture or scratch. The result is a local inflammation similar to small-pox, attended with an eruption and a fever, generally milder in form than small-pox acquired by breathing an atmosphere contaminated with the specific poison of the disease; and which thus passes through the mucous membrane to infect the blood. This is called the “natural way” of contracting small-pox; and the course of the disease so induced has been already noticed. For obvious reasons, the operation of *inoculating* the poison of small-pox has been rendered illegal in this country; and the practice of vaccination has been attempted to be enforced by law. What remains to be said about *inoculation* will be considered under the next topic.

* The average number of second attacks of small-pox seem to be one *per cent.* (R. Acad. of Med., Marseilles, 1828; and *B. and F. Med.-Chir. Review*, Jan., 1848, p. 74).

COW-POX.

LATIN, *Vaccinia*; FRENCH, *Vaccine*; GERMAN, *Kuhpocken*; ITALIAN, *Vaccinia*.

Definition.—*Cow-pox is the product of a specific and palpable morbid poison, which is reproduced and multiplied during the course of the malady in the cow or in the human being. After a definite period of incubation (from the time that the specific virus is artificially implanted, or communicated by impalpable emanations or effluvia in the "natural way") specific pimples form upon some part of the skin, which pass through the stages of vesicle, pustule, scab, and desiccation. During the maturation of these specific pimples the adjoining lymphatic glands swell; a febrile state is induced, denoted by increase of temperature, constitutional disturbance of functions, acceleration of the pulse (which, to a certain extent, has been observed to continue persistent in some cases); and a general lichenous, roseolar, or vesicular eruption makes its appearance on the trunk and limbs. The disease runs a definite course, affords immunity from another attack (for a considerable time at least), and exercises (during that period) a protective influence from human variola.*

Pathology and Symptoms.—The importance of a comprehensive knowledge of the pathology of variolous diseases generally, and of cow-pox in particular, lies in the relations of this latter disease to small-pox and to vaccination. Dr. Jenner named the disease "*variola vaccinae*," implying thereby that one genus at least of the animal creation is liable to a disease of a kindred nature with that which attacks man. The disease in the cow was observed to be generally mild; in man it was observed to be most pestilential and fatal. It was observed, also, that the disease was communicable from the cow to man, and that persons so affected were protected from subsequent attacks alike of small-pox and of cow-pox. Dr. Jenner believed that the two diseases were in reality identical. It has now been shown by unquestionable evidence that cattle and horses have for centuries been known to be affected with a species of small-pox or variola. Every different writer who has seen the disease has given it a similar name. Previous to 1745 it was known and described in Italy (FRACASTORIUS, LANCISI, RAMAZINI) as a malignant disease which destroyed cattle almost as extensively as small-pox did the

human race. It was first observed in this country in 1745, and again, in 1770, it appeared among the horned cattle with so much severity that His Majesty George III., in his speech from the throne, at the opening of Parliament on the 9th of January of that year, called upon the Houses of Parliament to take the subject into their serious consideration. The disease continued with more or less violence till 1780; and it was no doubt the expiring embers of this epizootic which Dr. Jenner found in Gloucester, and made the basis of his investigations during that and subsequent years. Dr. Layard described the disease amongst the cattle in England, in that year, in a paper communicated to the Royal Society; and he mentions that inoculation from cow to cow was successfully practised, to mitigate the severity of the disease; just as Mr. Simonds, of the Veterinary College, London, in 1862, successfully practised inoculation of the variolous disease from sheep to sheep, or lamb to lamb, throughout the counties of Wilts, Hants, and Dorset, when *ovine small-pox* was epidemic. The great increase of mortality from small-pox among human beings which occurred during the latter part of the last century is a fact of some importance in connection with the epizootic disease; for at other times and places it has been observed that when the cattle were scourged by the variolous disease, mankind were in like manner great sufferers from a similar epidemic. In the interesting lectures "Introductory to the Study of Fever," by Dr. Andrew Anderson, of Glasgow, we are told that while small-pox was raging with great violence at St. Jago, on the west coast of New Granada—to which a town named David, in Chiriqui, was situated about sixty or seventy miles to leeward—a few days (four or five) before the disease appeared in this latter town the small-pox had attacked and destroyed many monkeys in the forest. Dying and dead monkeys were seen on the ground covered with the perfect pustules of small-pox; and several sick monkeys were seen on the trees, moping or moving about in a sickly manner. In the course of a fortnight one-half of the inhabitants of the town of David were stricken with small-pox (ANDERSON, p. 70).

It is also within the experience of many medical men, that during the prevalence of small-pox, cattle are apt to become affected with cow-pox. Horses, as well as cows and sheep, are liable to the affection; and the countries where the disease of late years has been found are those where it has formerly been

known to have existed among cows or horses in its most virulent form. During the epidemics of small-pox previous to 1840 the variolous affections among the cows of the country were more observed than at any period for many years. In the dairies of Suffolk, of Gloucestershire, Dorsetshire, and Buckinghamshire, the disease has prevailed not only during epidemic small-pox, but when no cases of variola were known to exist in the immediate neighbourhood. There are good grounds for the belief that the impalpable emanations—the specific effluvia—from cases of small-pox in human beings, have been sufficient to communicate the variolous disease to cows. Mr. Ceely gives a most interesting history of such an occurrence in the tenth volume of the *Transactions of the Provincial Medical and Surgical Association*. At the village of Oakley, about sixteen miles from the town of Aylesbury, small-pox had been epidemic from June to October, 1840. Two cottages, in which three persons resided during their illness, were situated on each side of a long narrow meadow, comprising scarcely two acres of pasture land. One of these three patients, though thickly covered with pustules of small-pox, was not confined to her bed after the full development of the eruption; but frequently crossed the meadow to visit the other patients—a woman and a child—the former of whom was in great danger, from the confluent malignant form of the disease, and died. According to custom, she was buried the same evening; but the intercourse between the cottages across the meadow was still continued. On the day following death the wearing apparel of the deceased, the bed-clothes and bedding of both patients, were exposed for purification on the hedges bounding the meadow; the chaff of the child's bed was thrown into the ditch; and the flock of the deceased woman's bed was strewed about on the grass over the meadow, where it was exposed and turned every night, and for several hours during the day. This purification of the clothes continued for eleven days. At that time eight milch cows and two young heifers (sturks) were turned into this meadow to graze. They entered it every morning for this purpose, and were driven from it every afternoon. Whenever the cows quitted the meadow the infected articles were again exposed on the hedges, and the flock of the bed was spread out on the grass, and repeatedly turned. These things remained till the morning, when the cows were re-admitted, and the contaminated articles were supposed to be withdrawn. It

appears, however, that the removal of the infected articles was not always accomplished so punctually as had been enjoined, so that, on one occasion at least, the cows were seen in the midst of them, and licking up the flock of the bed which lay on the grass. These cows were in perfect health when first put out to graze in this meadow; but in twelve or fourteen days *five* (out of the eight) milch cows appeared to have heat and tenderness of the teats. The teats became swollen, and small hard pimples could be distinctly felt upon them, as if imbedded in the skin. These pimples daily increased in magnitude and tenderness; and in a week or ten days they rose into *blisters* (vesicles), passing into brown or blackish scabs. When the teats were in this condition, and very tender, constitutional symptoms of ill-health became developed. Sudden *sinking* or loss of milk, drivelling of saliva from the mouth, frequent inflation and retraction of the cheeks, staring of the coat, "tucking up of the limbs," "sticking up of the back," and rapid loss of flesh were the appearances which even the peasants themselves were able to appreciate. By the middle of the third week the pustules were mature, and the crusts and loose cuticle began to be detached. The simultaneous occurrence of the disease on all the animals increases the probability of the operation of one common cause. The whole of the cows were certainly affected within less than three days of each other; and another circumstance requires particular notice, namely, the occurrence of the disease in a young heifer (*sturk*), to which of course the disease could not have been communicated by those casualties which commonly propagate the vaccine variola amongst milch cows. The cause which originated the disease amongst them at the same time affected the young heifer, which hitherto had not been considered liable to the vaccine disease, simply because no one had seen the animal affected by it. Now it is known, both in this country and in Germany, to be liable to the disease.

The proprietor of the animals referred to in this narrative had the disease communicated to himself. He had never suffered from small-pox nor the vaccine disease; and it was his own spontaneous conviction "*that his cows had been infected from human small-pox effluvia*," to which undoubtedly they had been exposed. He had not the remotest idea of the medical theories concerning the nature of the disease, and consequently had no

prepossession in favour of the opinion he thus spontaneously expressed. His cattle had hitherto been in good health, and no vaccine variola had been known in the vicinity.

Human small-pox has also been communicated to the cow by direct implantation of the specific virus from man. The efforts at first were numerous and unsatisfactory to inoculate directly the cow with human small-pox; and the experiment is said to have first succeeded at the Veterinary College in Berlin, so early as 1801. M. Viborg, of Copenhagen, about the same period communicated the disease to dogs, apes, and swine. In 1807 Gassner imparted the small-pox to the cow by inoculation. In 1830 or 1831 Dr. Sonderland, of Bemen, communicated the disease to cows, by simply covering the animals with sheets and blankets on which persons suffering from small-pox had lain. In 1836 Dr. Basil Thiele, of Kasan, in South Russia, successfully inoculated some cows on the udder with the *virus* of human small-pox. Vesicles were produced bearing all the characters of the true vaccine vesicle in those animals. The lymph so produced from the *variolation* of the cow continued to retain the specific properties of the vaccine variola throughout seventy-five successive transmissions in the human subject. In 1838 M. Thiele repeated this interesting experiment with a similar success. It would therefore seem that the constitution of the cow has the power of assimilating, of modifying, and of mitigating the human *variolous virus*, and of stamping it with the properties of the vaccine variola. Dr. Ceely, of Aylesbury, twice succeeded in accomplishing this object (so important pathologically), after many fruitless trials. The interesting papers by him in the eighth and tenth volumes, and the Reports of the Vaccination Section of the Provincial (now British) Medical Association in their *Proceedings* for 1839 and 1842, should be studied by every student of Medicine. The main points of the statement here given are taken from these sources. Very recently (1860) Martin inoculated some variolous matter, taken from a pock upon the body of a man who died of variola, into a cow's udder, and subsequently vaccinated about fifty persons with the matter derived from the cow. Most of those so inoculated were attacked with variola, and three died (*Boston Med. Journal*, 1860; *New Syden. Society Year-Book* for 1860). It would have been better, or at least more judicious, to have chosen a milder case than a fatal

one to have inoculated from. Mr. Ceely has also often communicated the vaccine disease from man back to the cow (*retrovaccination*, as it has been called); and he has observed that good human lymph, when *re-transmitted* in this manner, loses some portion of its activity. The phenomena appear later, smaller vesicles are produced, but ultimately, *after successive re-inoculations on man*, it regains its activity. Human small-pox has also been transmitted through the horse to the cow, and so to the child in the form of cow-pox (FLETCHER).

As the first origin of these specific poisons is as yet unknown, it cannot be now definitively determined whether man first had the disease communicated to him from the animal creation, or whether the lower animals, such as horses or oxen, had the disease communicated to them from man. The existence of small-pox in man is recorded in China as early as 1,122 years before Christ (MOORE). And it is certain that when variolous disease appears among the lower animals in a malignant form, it is capable of producing, by inoculation, a disease of similar severity in man, if he has not already suffered from a similar affection; and that the direct inoculation of the cow with human small-pox produces a mild and mitigated form of disease—that such disease being again reproduced in man by inoculation from the mitigated disease of the cow, accords entirely in its character, in its progress, and in its protecting influence with the *variola vaccinae*, as described by Dr. Jenner. These and similar facts seem to lead to the conclusion that small-pox and cow-pox are not dissimilar diseases, but are identical in their nature. 3 MB

There are some remarkable circumstances which must at once arrest the attention of the student who carefully studies the accounts given of the experiments on men and animals, from which many of these statements are deduced. *First*, There seems to have been great uncertainty and difficulty often attending the actual attempts to transfer the specific virus of these eruptive or variolous diseases from one animal to another. The very interesting experiments of Ceely, and of Thiele and others, demonstrate this in a remarkable manner. *Second*, These experiments show the marked improvement which sometimes takes place in the energy, and therefore in the quality of the specific virus, by subsequent removes or inoculations, in animals of the same kind, after the virus had been successfully implanted in one of them.

This energy and improved quality was shown in the more perfect development of vesicles, and in the more active manifestations of the primary and secondary symptoms. The subsequent inoculations of such improved lymph seem to produce less severe and less dangerous *local* results—the virus seems less acrid, less virulent, and less mischievous—having apparently acquired increased specific activity combined with mildness of action, and a greater susceptibility of transmission from one animal to another of the same kind.

Keeping, therefore, these facts in view, the history of the remarkable epizootic of *variola ovinae* which made its appearance in August, 1862, in some of the largest breeding flocks of sheep in the West of England, is of great interest to the Pathologist. This variolous disease of the sheep allies itself very closely with small-pox in man, with cow-pox amongst cattle, and with the vesicular eruptive diseases of the horse, but undoubtedly modified by the constitution of the sheep, just as the variola of man is known to be modified or transformed by the constitution of the cow into the *variola vaccinae* of that animal.

The ovine variola is known as the *clavelée* of the French; and although this kind of *rot* was not observed in this country till 1847, when it was imported from Spain, yet it is a disease by no means uncommon as an epizootic in the flocks of Italy, France, and Moravia. In 1803 the mortality was considerable in Moravia; but by a timely inoculation with the *virus* of the disease, the remaining part of affected herds were preserved. The artificially affected animals seemed to pass through a milder disease. To this kind of inoculation the name of "*clavelisation*" has been given, from *clavelée*, the French word for the *tag-sore* or *rot*. This variolous disease in sheep assumes one of two forms, namely, —(1.) A virulent or malignant form; and (2.) A benign form. The virulent form (which would seem to have been the form epizootic in England) never produces pustules; and specific virus for safe inoculation (*clavelisation*) can only be got from the benign form of the disease. When the disease is virulent, the sheep lose their eyes, their wool falls off, and their skin cracks in a zig-zag manner. Their nostrils are so full of a fetid discharge that the shepherds are under the necessity of constantly syringing them with medicated lotions, to prevent suffocation. When the disease is benign, genuine pustules form, and every pustule, after

the scab falls off, leaves a cicatrix in the form of a pit. On this cicatrix the wool never grows again. Hence it can always be told what sheep have undergone the variolous disease, as easily as it can be seen that a human being has had small-pox. WRB

The prevention or mitigation of this disease among sheep is a most important object in a sanitary point of view.

In 1803 Dr. De Carro, of Vienna, tried the effects of the inoculation of *variola vaccinae*, but without success. The inoculation only produced small local sores. It is said, also, that this sheep-pox cannot be communicated directly to the cow, nor to children (CEELY, SIMONDS). Other observers state, however, that it is so communicable, and that *ovination* is protective against small-pox (SACCHO). The *ovination*, or inoculation of the disease from sheep to sheep, was first proposed by Chalette in 1762, and has been yearly practised since that time in many parts of Italy, Prussia, Austria, and France. The practice of inoculation from sheep to sheep was practised in England by Professor Simonds during the recent epidemic. Results accrue to sheep, from the communication of the disease to them by inoculation, not less beneficial (compared with the fatal effects which followed when they became affected with the disease in the "*natural way*") than the beneficial effects that accrued to man when small-pox was communicated to him by inoculation—as it *rightly* was—before the protective powers of vaccination were known.

The ovine variola has been ascribed by some farmers to the communication of the virus from an eruptive disease of the horse (DE CARRO, RING). Fontan relates that some mares being affected with a pustular eruption, the matter from the pustules was inoculated on the teat of a cow, where it produced several fine pustules. From these several infants were vaccinated, with the result of producing perfectly characteristic vaccine vesicles. Thirty infants have been vaccinated from this source at Toulouse, and in all the result has been most satisfactory (*L'Union Méd.*, 1860; *New Syden. Society Year-Book*, 1860). If this can be definitively established, then the successful inoculation of some animal, *other than the sheep*, with the virus from the specific eruptive disease of the horse may give such energy and, at the same time, mildness to the morbid poison, by subsequent removes, that the implantation of the new virus (*equination*) may perhaps be followed by the same beneficial

results to sheep, in respect of the malignant variolous disease to which they are liable, that *vaccination* has conferred on man in respect of small-pox. The question, then, at once suggests itself: "Has human small-pox ever been communicated to sheep, with the view of obtaining a modified lymph which may confer protection on them from the variolous disease to which they are liable?"

From analogy, may we not indulge the hope that the practice of inoculating sheep from the small-pox of man might induce as mild and modified a disease in them, and prove as protective to them, as vaccine variola, through vaccination, has been to man? Or, having communicated the human variola to cows (as the experiments of Ceely and Thiele demonstrate that such may be effected), might not sheep be tried with the resulting virus as a protective agent? The vesicular eruptive diseases of dogs, as well as of horses, should be similarly inquired into and experimented with, seeing that dogs are so much associated with sheep.

The outbreaks of the variolous diseases amongst cattle and sheep seem to follow similar inexplicable paths to those which small-pox amongst human beings is observed to follow. Occasionally the disease is *epizoötic* (equivalent to *epidemic* amongst men), or prevalent at the same time in several farms at no great distance. Cases spring up like small-pox, now and then, which appear to be solitary, and the source of which cannot be traced. It is rare indeed that the solitary cases of small-pox in human beings can be traced to a communicating source. In oxen it may be seen sometimes at contiguous farms; at other times, one or two farms, apparently similarly circumstanced amidst the prevailing disease, entirely escape its visitation. Sometimes it is introduced into a dairy by recently purchased cows. On the other hand it has been undoubtedly communicated to cows from the vesicular disease of the horse, through the hands of the common attendant on both animals. There can be no doubt, also, that the disease often exists, although it is not observed; for the disease being mild, and the tempers of the animals good, little notice is taken of tenderness in milking, and so the existence of disease escapes detection.

There are *spurious* forms of the disease, which it is very necessary to be able to distinguish.

In the true cow-pox there is very slight manifestation of fever or constitutional disturbance. The secretion of milk may

diminish; but the animal continues to feed and to graze very much as usual. The local affection may be so mild that a single vesicle only may appear upon the udder of the cow; but where the udder is voluminous, flabby, and pendulous, and uncovered with hair, with a corrugated, thin, or fissured skin, then there is apt to be a copious eruption. The disease is very readily propagated from cow to cow by the milkers; and it is also said to be communicated in the natural way.

The local symptoms of the natural disease are evinced by heat and tenderness of the teats and udder for three or four days, followed by irregularity and pimply hardness of the surface, especially about the bases of the teats and the adjoining part of the udder. The pimples assume a red hue when about the size of a vetch or pea, and are quite hard. In three or four days more they increase to the size of a horse bean, milking becomes painful to the animal, and the pimples become vesicles, which are then apt to be broken by the hands of the milkers, giving rise to troublesome and dangerous sores on the udder and teats of the cow, and communicating the disease to the milker, if he is not already protected by having had the disease before. If the vesicle remains unbroken it becomes a globular, oval, and ultimately a pointed (acuminated) pustule. A central depression, with a marginal induration, is the form ultimately assumed; and when punctured towards the centre, the vesicles yield a more or less viscid amber-coloured fluid. Dark-brown or black, solid, uniform crusts eventually form on the site of the vesicles. Some of these crusts may be seen semi-detached, others entirely so, and exposing a raw surface with a slight central slough. The forms of the crusts are either circular or oval, some flatter, and others unguiform, some thin, and more or less translucent. These varied appearances are seen in all stages at the same time, indicating the formation of new crops of vesicles at different periods. The period of incubation, after casual communication of the disease, seems to be from six to nine days, although it is said pimples may be felt under the cuticle about the fifth day. When the vesicles are fully mature, they may measure from eight to ten lines in the largest diameter; the centre and edges of the intumescent margin being of a deep blue or slate colour, and the surrounding areola of a pale rose colour, and seldom more than four or five lines in depth, the integuments under it being deeply

indurated. The lymph contained in the vesicle is now so copious that the cuticle over the central depression appears raised up by it, and so gives rise to a globular or cone-like vesicle; or it spontaneously ruptures, when the lymph freely flows out, and concretes into a clear amber-coloured crust or scab.

If undisturbed, this crust or scab gradually becomes thicker, darker, and more compact, till the thirteenth or fourteenth day, and spontaneously separates about the twentieth or twenty-third day. A cicatrix or pit is thus left, which is shallow, smooth, oval, or circular, of a pale rose or whitish colour, with some traces of induration surrounding it. The anatomical structure of the vesicle seems to be precisely similar to that of small-pox in man, as shown now by many observers (GENDRIN, CEELY, and others). The cow—like children and the young of other animals, particularly high-bred dogs—is subject to a purely vesicular eruption, which makes its appearance about the ninth or tenth day of the vaccine disease. The vesicles of this eruption, within twenty-four hours, contain a pellucid serous fluid, raising the epidermis. On the following day they become turbid, the cuticle collapses or bursts, and a thin, brittle, flimsy crust forms, and speedily falls off. Successive crops continue to form and desiccate for three or four weeks.

Primary Vaccine Lymph.—To procure *primary* liquid vaccine lymph direct from the cow, in a condition fit for use, is a task of no ordinary difficulty. *Primary* crusts should be sought for on the lower part of the udder and around the base of the teats; and during a search for these it is not improbable that smaller vesicles of later growth may be found to yield efficient lymph. The best lymph is to be obtained from perfect vesicles, before they begin to point. After this period it is less to be depended on, particularly if very abundant, thin, or discoloured. Pointed vesicles, when broken by violence, are rarely to be relied on. Entire unpointed vesicles, or vesicles with central crusts, should be sought for on parts where they are least exposed to injury—namely, on the lower and naked parts of the udder and adjoining bases of the teats. It is impossible to exercise too much delicacy in the proceeding. The puncture to liberate the lymph should be made with a sharp lancet as near the centre of the vesicle as possible; and the epidermis may be gently raised to a moderate extent around the discoloured or most depressed part. Slight

pressure with the blade of the lancet, or between the thumb and finger, will enable the operator to charge a few points or capillary tubes with the slowly exuding lymph. Punctures at the elevated and indurated margin of the vesicle are utterly useless. They only give vent to blood. Vesicles on which the central crust has begun to form are the most productive, particularly if the crust be small and the margin of the vesicle be tender, hot, and tumid; and small superficial vesicles are often more yielding than contiguous larger vesicles, which are more deeply seated or confluent.

Useful substitutes for liquid lymph, capable of communicating the vaccine disease, are—(1.) Amorphous masses of concrete lymph, found upon or in close proximity to broken vesicles. They ought to be colourless, like crystals of white sugar-candy; or of a light amber hue, resembling fragments of barley-sugar. (2.) Central crusts, irregular, rough, and more or less conical; the more transparent and nearer a dark-brown hue the better. (3.) Vesicular crusts or desiccated vesicles. These crusts should be carefully removed by the milkers before they are casually removed or spontaneously fall; and those only of primary formation, which are as it were the mould of a vesicle, of a dark-brown translucent appearance, should be retained. These three dry conditions of the specific vaccine virus may be reduced to a liquid state at any time for use. Glycerine is said to be the best solvent for such solid conditions of the lymph, which ought to be reduced to powder before the glycerine is added (Collins, *Boston Med. and Surg. Journal*, 1858).

Vaccination.—It is now (1864) at least sixty-four years since Jenner first promulgated his discovery to the world, that the eruptive vesicular disease which has now been described as occurring on the udders and teats of the cow, and which he named the *cow-pox*, was communicable directly to the human being, and thus conferred protection from the small-pox, so fatal to man. The operation for thus ingrafting the cow-pox on the human being has been named “VACCINATION;” and its discovery still remains one of the most interesting facts in the history of Medical Science. When an obscure apprentice with a surgeon at Sodbury, near Bristol, Dr. Jenner first caught a glimpse of this great truth, which he thoroughly investigated amongst the expiring embers of that epizootic disease which laid waste the

herds of this country towards the end of the last century. He did not suffer the spark to be lost in the flame it had served to kindle. Amongst the gossip of the cow-herds he had heard of the vague, obscure, but popular belief regarding the possible communication of cow-pox to the milkers of the cows, and the protection from small-pox which the cow-pox conferred—a belief which undoubtedly prevailed in the rural districts of Gloucestershire, and, at the same time, curiously enough, on the continent of Europe, in some districts round Göttingen. These things Jenner mentioned to the famous John Hunter, at the time he was an apprentice to that eminent surgeon; but John Hunter, otherwise sagacious and far-sighted, pooh-poohed the notion as vague and improbable. Nevertheless, Jenner had determined to examine into the truth of the tradition; and he commenced his earnest and painstaking investigations as soon as he had established himself as a surgeon at Berkeley, in Gloucestershire. In June, 1798, he published his observations in the form of a thin quarto, of scarcely more than seventy pages, dedicating it to his friend, the late celebrated Dr. Parry, of Bath. Jenner seems to have felt almost a holy reliance in the truth of his great discovery; and in the face of much foolish opposition he modestly continued to prosecute his inquiry, “encouraged,” as he said, “by the hope of its becoming beneficial to mankind.” Its importance to the welfare of the human race has since been clearly demonstrated; and the acute observation of Jenner himself has been abundantly fulfilled—namely, that the keenest of all arguments for or against the practice of vaccination will be *those which are engraved with the point of the lancet*. We have, indeed, in this country, paid but tardy homage to his memory; nevertheless, he has imprinted for himself imperishable “footprints on the sands of time,” which wave after wave of scientific research appears only to make more distinct. He has not only pointed out the means of subduing a loathsome disease, but the health of all civilized communities has improved, and, in proportion as vaccination has been efficiently carried on, the frequency of epidemics has been diminished, and the duration of human life has been extended.

The subject of vaccination is one which demands a careful study, alike in its pathological and in its sanitary relations. Questions of great national importance are concerned; and the following account is mainly given from a notice of the subject,

written by the author, in the pages of the *Medico-Chirurgical Review* for 1857:—

In 1841 the Vaccination Act was passed, which rightly made the practice of *inoculation unlawful*. In 1853 another Act was passed, with the view of rendering the practice of *vaccination compulsory*,—an Act which is known as Lord Lyttleton's Vaccination Act. During the interval between the first and second reading of the Bill in the House of Commons, "The Small-Pox and Vaccination Committee of the Epidemiological Society completed a report on the prevalence and mortality of small-pox, and of the means taken to guard against it through vaccination." The conclusions they arrived at were deduced from the largest and most accurate mass of statistical evidence which had ever been brought to bear upon the question, and were eminently calculated to encourage Her Majesty's Ministers to pass an efficient measure to *compel* vaccination. A most valuable pamphlet was afterwards published by Dr. Seaton, which demonstrates the truth in a still more forcible manner, as to the protecting and modifying influence of vaccination in small-pox. To this belief, indeed, the general assent of the medical profession appears to have been given at least fifty years ago. *Then*, it would seem to have been all but unanimous; and *now*, one would think, at first sight, that it were almost an insult to human understanding to be obliged to collect statistics to *prove* that vaccination confers a large exemption from attacks of small-pox, and almost absolute security against death from that disease. But so it is, and independently of the information which such statistical inquiry is calculated to convey to those who advise our Lawgivers and Public Administrators, the inquiry is eminently useful in relation to everything which bears on the nature of vaccine and variolous disease. The general ignorance of the community, especially of the lower orders, as to the aim and object of vaccination, is lamentably great, and has still to be overcome. Moreover, the highest medical authorities of late years recommend that all views and facts put forward as objections to vaccination should be vigorously inquired into, and that there should be published from time to time a true account of such inquiries, with an elucidation of what has seemed doubtful and contradictory (SIGMUND, ALISON).

It is now well known that Lord Lyttleton's Vaccination Act

(1853) has proved but a very imperfect measure—a piece of legislation which has fallen very far short of accomplishing all that is yet required. The inefficiency and imperfect working of the Act has been fully shown,—(1.) In the Reports of the Registrar-General for 1854; (2.) By the medical profession generally; (3.) By the medical registrars in particular; (4.) By the public, as expressed now and again in the newspapers of the day. To this state of things we owe a most valuable work on vaccination, written by the indefatigable medical officer (JOHN SIMON, F.R.S.) of the then (1857) General Board of Health. The aim of this publication was to lay before the Board such medical facts and considerations as might assist in estimating the hygienic value of vaccination, and the strength of any objections which may have been alleged against its general adoption. This work is especially valuable, because it brings together a body of evidence down to the day of its publication—evidence of a pathological and statistical kind—such evidence as Jenner would have rejoiced to see—records which have been engraved by the lancet's point. But evidence of the inefficiency of the Vaccination Act of 1853 still continues to be apparent, as may be seen from the yearly reports of the medical officer of the Privy Council. During three or four years previous to 1860, Mr. Simon writes, that "Sometimes in one set of places, and sometimes in another, there have been occurring, almost generally throughout England, epidemics of small-pox more or less considerable." To such an extent has this been the case, that, in 1860, the Lords of Her Majesty's Most Honourable Privy Council deemed it necessary, with reference to local outbreaks of this disease, to enter into correspondence with the authorities responsible for public vaccination in several unions of England where small-pox had been prevalent. Some of these districts were specially visited by competent medical officers, and in two adjoining Devonshire Unions it was ascertained that the *diffusion of small-pox* had actually been to some extent *wilfully promoted by the illegal practice of INOCULATION!* Alarm was of course justly excited among educated persons in the endangered places, by the knowledge that this offence was being committed; and in one case, where there was reason to believe that *inoculation had been the cause of death*, Secretary Sir George C. Lewis offered a reward of £50, to be paid to any person not actually concerned in the

offence, who would give information and evidence leading to a conviction of the offender.

The unsatisfactory working of the Vaccination Act of 1853, now amply proven, has led to the promulgation of an Order of the Privy Council (of date December 1, 1859), for the improvement of public vaccination. Their Lordships have seen fit to direct the commencement of a *systematic inspection*, with reference especially to the operation of vaccination, and its efficiency in Unions where the amount of infantine vaccinations, compared with the number of births, appeared to be especially low. These inquiries continue to show that the present law, "to extend and make compulsory the practice of vaccination," is so imperfect as to be almost inoperative; and the *systematic inspections* instituted seem to have been so useful in promoting vaccinations that their Lordships propose to continue them throughout all the Unions of England.

The present position of our knowledge regarding vaccination is based upon evidence which demonstrates,—(1.) The *protective influence of vaccination*; and (2.) The causes which have combined to *impair its protective power*. Of these in their order.

I. *Of the Protection conferred by Vaccination—its Nature, and the Evidence of its Existence.*

The main features of the reports and works already mentioned amply illustrate how small-pox diminishes in its mortality *in proportion as EFFICIENT measures are adopted to insure PERFECT vaccination*. To demonstrate this statement, the progress of vaccination in Great Britain and in Germany has been compared as to its influence on mortality generally; and more particularly, it has been shown, by comparing the statistics of vaccination from various German states with similar statistics from different districts in Great Britain and Ireland, that where vaccination is most perfectly carried out, small-pox is least mortal. The following are the general results which the Committee of the Epidemiological Society arrived at:—

1. To prove the influence of vaccination in England, it is shown that out of every 1,000 deaths in the half-century from 1750 to 1800 there were 96 deaths from small-pox; and out of every 1,000 deaths in the half-century from 1800 to 1850 there were only 35 deaths from small-pox.

2. To prove the influence of vaccination on the Continent, it is

shown that in various German states sufficient evidence can be obtained to show that out of every 1,000 deaths *before vaccination was used*, 66·5 were deaths from small-pox; but that out of every 1,000 deaths *after vaccination* came into use, the deaths from small-pox were only 7·26.

3. To prove that in countries where vaccination is most perfectly carried out small-pox is least mortal, it is shown that—

(a.) In this country, where vaccination has been voluntary, and frequently neglected, the deaths from all causes being 1,000, the deaths from small-pox were as follows:—

London, ...	16	Edinburgh, ...	19·4
Birmingham, ...	16·6	Glasgow,*	36
Leeds, ...	17·5	Galway,*	35
England and Wales, ...	21·9	Limerick,*	41
Perth, ...	25	Dublin, ...	25·6
Paisley, ...	18	Connaught,*	60
All Ireland, ...		49	

(b.) In other countries, where vaccination has been more or less compulsory, the deaths from all causes being 1,000, the deaths from small-pox were as follows:—

Westphalia, ...	6	Bohemia, ...	2
Saxony, ...	8·33	Lombardy, ...	2
Rhenish Provinces, ...	3·7	Venice, ...	2·2
Pomerania, ...	5·25	Sweden, ...	2·7
Lower Austria, ...	6	Bavaria, ...	4

Evidence corroborative of these results has been adduced by Dr. Balfour from the records of the Army and Navy Medical Departments, where every soldier or sailor is protected by vaccination, if he has not previously suffered from *cow-pox* or *small-pox*.

1. For twenty years, namely, from 1817 to 1836 inclusive, it is shown that in Dragoon Regiments and Guards, with an aggregate strength during that period of 44,611 men, and a total mortality of 627, only *three* deaths were from small-pox.

* With regard to the high rate of small-pox mortality in the towns marked by the asterisks, it was clearly shown by Dr. Stark, in Edinburgh, and by Dr. J. C. Steele (the present Medical Superintendent of Guy's Hospital in London), that such mortality was due to the neglect of vaccination. Dr. Steele, then resident in Glasgow Infirmary, called attention to the great increase of small-pox in Glasgow, as mainly coming from the Highland and Irish population, among whom vaccination was rare. Dr. Stark showed that more than eighty *per cent.* of all the deaths from small-pox happened in children under *five* years of age.

2. Among the troops at Gibraltar, the aggregate strength being 44,611 men during that period, and a total mortality of 1,291, only *one* death from small-pox occurred.

3. In the West Indies several epidemics of small-pox prevailed during the period, but there were *no deaths* either among the British or white troops, of whom the aggregate strength was 86,661, and with a total mortality of 6,803. Among the black troops on the same station, with an aggregate strength of 40,934, and a mortality of 1,645, there was not *one* case of small-pox.

4. At Bermuda, Nova Scotia, New Brunswick, Cape of Good Hope, and the Mauritius, not a death from small-pox occurred during the twenty years mentioned; and the white troops of Western Africa wholly escaped this disease, while the black unprotected population were dying by hundreds.

5. In Malta, from 1818 to 1838 inclusive, the aggregate strength of the British troops being 40,826 during these twenty years, and the total mortality 665, only *two* deaths were from small-pox. This is the more remarkable, inasmuch as small-pox raged as an epidemic all over the island in 1830, and again in 1838, destroying 1,169 persons. In 1830 there died of small-pox 1,048, out of a total mortality of 3,407; and in 1838 there were 121 deaths from small-pox, out of a total mortality of 2,583. The disease was first introduced by His Majesty's ship "Asia." The mortality among those "*not vaccinated*" was 1 in 4·7; among those "*supposed to have been vaccinated*" it was 1 in 23·4; and, lastly, among those attacked a second time by small-pox the mortality was 1 in 10·8. The native population of Malta in 1830 was estimated at 100,839 persons, amongst whom it appears that 1 in every 12·1 persons was attacked with the disease, and 1 in every 85 persons died. Amongst the military, including wives and children, the proportion attacked was 1 in 188, and the mortality only 1 in 682.

6. The most important observations are those made at the Royal Military Asylum, in which the prevalence of variola among a *vaccinated* and *unvaccinated* population, at the same ages and in exactly similar circumstances, has been traced out by Dr. Balfour (See *Med.-Chir. Trans.* for 1852).

One very obviously beneficial result of vaccination has not been so much appreciated and noticed as it ought to be—namely, that while the epidemic influence of small-pox greatly *increased*

during the practice of *inoculation*, it has greatly *diminished* since vaccination has been adopted. Dr. Hebra, of Vienna, alludes to the fact, and incidentally remarks, "That epidemics of small-pox have been more rare, and are less malignant, since the introduction of vaccination." Definite data are to be gathered from the various reports already mentioned, which fully bear out the belief. The results may be stated as follows:—

1. During ninety-one years previous to *inoculation*, there are on record 65 distinct and well-marked epidemics; which is equal to a ratio of 71·4 epidemics in 100 years.

2. During sixty-three years in which *inoculation* was practised, and that to a very great extent, there were 53 distinct and well-marked epidemics; which is equal to a ratio of 84 epidemics in 100 years.

3. During the fifty-five years since *vaccination* has been mainly practised, there have been 12 distinct and well-marked epidemics of small-pox; which is equal to a ratio of 24 epidemics in 100 years.

This kind of testimony is greatly enhanced by the fact, that epidemics never occur in the army or navy of our own country, nor in those countries where the soldiers and seamen are efficiently protected by vaccination. The details given with reference to the two Malta epidemics in 1830 and 1838 afford a striking proof of the protective power of vaccination when tested by epidemic influence; and there are records of the Danish army and navy having altogether escaped during several epidemics of small-pox in Denmark.

There is still another way in which the protective power of vaccination makes itself manifest—namely, by the *mildness of the disease in the vaccinated compared with the unvaccinated, and the almost absolute security against death from small-pox which PERFECT vaccination confers*. With few exceptions, this appears to be the universal belief of the medical profession. At various times the opinions of large numbers of medical men have been specially asked for and obtained on this point. Three distinct and very comprehensive "*polls*" may be referred to, namely,—(1.) That by the College of Physicians in London, eight years after vaccination had been adopted. (2.) An almost national "*poll*" taken by the Epidemiological Society of London about the years 1852 and 1853. The written opinions of nearly

2 per cent mortality among well vaccinated
Phthisis & Typhoid fever dim'd also

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two thousand medical men in this kingdom, as well as Bombay, Bengal, the Mauritius, the West Indies, and various other places, were here expressed; and they concurred in confirming the belief in the protective and modifying influence of vaccination in small-pox. (3.) A very extensive "poll," of which a list is published by Mr. Simon, comprehends not only members of the medical profession generally, but also the members of the Medical Department of the Army and Navy, together with the opinions of foreign governments.

These "polls" may be held as completely decisive of the question, really practically decided in the affirmative fifty years ago. From such evidence the inference is so inevitable, "that he who disputes it is equally unreasonable as he who opposes in like manner any proposition in Euclid" (ALISON).

The actual *extent of the security against death from small-pox* enjoyed by vaccinated compared with unvaccinated persons, has been calculated by Mr. Simon from various sources; and it appears that the death-rate from small-pox amongst the vaccinated varies from an inappreciably small mortality to $12\frac{1}{2}$ per cent.; that amongst the unprotected the death-rate from small-pox ranges from $14\frac{1}{2}$ to $53\frac{1}{2}$ per cent.

The average *per centage* mortality from small-pox, stated by Mr. Marson to occur amongst the vaccinated, is 5.24; but when vaccination is known to have been perfectly performed, as shown by the cicatrices, the mortality is uniformly found to be reduced to less than *half of one per cent.*

Another very important and most interesting phase in which vaccination may be viewed as exercising a protecting influence *indirectly* over the health of the community, may be studied in those statistics which show that the *general death-rates from other diseases* have diminished, and more especially as regards *scrofulous diseases*, since vaccination became more universal. To Dr. Greenhow and to Dr. Farr in this country, and to the Statists of Sweden, we are indebted for any accurate knowledge existing on this subject. Not only has the grand total of the death-rates been diminished, but the death-rates of two special classes of diseases have diminished in a remarkable degree. These are,—(1.) Those of the tubercular order of the constitutional diseases—namely, scrofulous affections, including phthisis or pulmonary tuberculosis; and (2.) The continued fevers, especially typhoid

fever—diseases belonging to the miasmatic order of the Zymotic class.

Another kind of historical evidence bearing out the protective influence of vaccination is to be seen by comparing the advertisements of old newspapers, especially those during the last portion of the seventeenth and early portion of the eighteenth century, with similar advertisements of the present day. At the former period the ravages of small-pox upon the population were beyond conception, testifying to the wide-spread epidemic influence. The description of every man or woman, as exhibited in "the villainous portraits of the Hue and Cry," showed them to have been more or less marked with small-pox, or "speckled with *pock-holes*." Now-a-days it is the exception, and not the rule, to see such pits and scars amongst the population (*Quarterly Review*, July, 1855).

It is thus clearly demonstrated how vaccination has thrown the *ægis* of protection over the world; and how ample, how great, and how efficient that protection may be. It has been shown to diminish mortality generally, and the mortality from small-pox in particular, both in civil and in military life, at home and abroad, and just in proportion as it is *efficiently* performed. It has been shown to diminish the epidemic influence; it has been shown to preserve the good looks of the people; it has been shown that it tends to render small-pox a mild disease compared with the same disease in the unprotected; it confers an almost absolute security against death from small-pox; and, lastly, it has been shown to exercise a protecting influence over the health of the community generally. On the other hand, it is no less amply proven that "wheresoever vaccination falls into neglect, small-pox tends to become again the same frightful pestilence it was in the days before Jenner's discovery; that wheresoever vaccination is universally and properly performed, small-pox tends to be of as little effect as any extinct epidemic of the middle ages" (SIMON). Moreover, it has been clearly shown by the *systematic inspections* instituted by Her Majesty's Privy Council, under the direction of Mr. Simon, that it is hopeless to expect to be free from fatal epidemics of small-pox, of greater or less extent, so long as unvaccinated children are allowed to accumulate as they have been found to do. There is therefore the greatest necessity for vigilance on the part of every intelligent

member of the community to prevent any re-accumulation of unvaccinated persons.

Four conditions are absolutely necessary to be efficiently carried out before we can hope to see small-pox eradicated through vaccination, and the aim of Jenner accomplished. These are,—(1.) The vaccination of every child must be made compulsory within a certain time after birth. (2.) *Systematic inspections* of two kinds must be constantly and periodically made by competent persons—namely, one to ascertain as to the effectual performance of the operation, as evinced by the kind of cicatrix visible. This inspection may be most conveniently made in public and private schools. Another inspection should have for its object to ascertain the numbers vaccinated within a certain territory compared with the numbers born in the same place. (3.) Every attempt at *variolous inoculation* ought to be made a penal offence. (4.) Every case of small-pox ought to be treated in strict seclusion, and be as completely as possible isolated, following out all the directions given with reference to the management of epidemics at page 226, as are applicable to the case. A quarantine regulation to enforce segregation of the sick from small-pox is of far more importance in this country than for yellow fever, which does not find a *habitat* with us. We come now to consider—

II. *How the Protective Influence of Vaccination has been Impaired.*

Since vaccination has been generally practised it has now and then seemed apparent that “*the protective power of vaccination becomes gradually weaker, and at length dies out in the individual.*” The works and reports which have been mentioned seem to demonstrate the truth of this statement; but in justice to Dr. Seaton (one of the greatest authorities on the subject) it must be stated that he does not subscribe to this belief. Indeed, in his last Report (*Appendix to Public Health Report for 1861*, p. 64), he states that where uniform care in the selection of lymph, and in the performance of the operation, was practised, the results did not favour the hypothesis that there had been any necessary deterioration of the lymph. He has seen several cicatrices, the results of the vaccinations of Dr. Jenner and Dr. Walker; but the work of the vaccinators to whom he refers (and mentions as having bestowed great care in the selection of

their lymph and in the performance of the operation) will bear comparison with the results obtained by Dr. Jenner and Dr. Walker.

In 1809 Mr. Brown, of Musselburgh, near Edinburgh, published the opinion that the prophylactic virtue of cow-pox diminished as the time from vaccination increased. In 1818 and 1819 small-pox prevailed in Scotland as an epidemic, and many vaccinated persons passed through a mild form of variola. The terms "modified small-pox" and "varioloid disease" about this time came into general use; and two classic monographs on the subject made their appearance, one by Dr. Monro, in 1818, and another by Dr. John Thomson, of Edinburgh, in 1820. Dr. Copland also writes that he saw and described, as early as 1823, small-pox as it affected members of the same family at different periods after vaccination, and in young persons who had been vaccinated only ten or eleven years. Contrasting such cases, he found that the severity and fully developed condition of small-pox was generally in proportion to the length of time which had elapsed from vaccination (*Dictionary of Practical Medicine*, Art. "Small-pox," p. 815).

Again, from the evidence contained in the bills of mortality of 1825—from the experience of epidemics of small-pox in France and Italy, in 1826, 1827, and 1829—from the experience of the epidemics of small-pox in Ceylon in 1833 and 1834—and from the admissions into the London Small-pox Hospital in 1838, it has been rendered obvious that the susceptibility to small-pox, which in vaccinated persons is destroyed for some years, returns with advancing age, and becomes greater as life advances.

Some of the phenomena, also, which the practice of vaccination itself has made known to us tend to establish the doctrine of a gradual impairment of vaccine protection, due to *lapse of time*, and as a result of *physiological changes in the healthy body*. This is especially indicated by the fact, that in proportion (undetermined) to the distance of time that has elapsed from the first implanting of the vaccine virus, so is the better development of the vaccine vesicle produced by re-vaccination. It has been shown, however, from a careful analysis of cases, that the lesser protectedness of certain vaccinated persons bears at least some proportion to the number of years which had elapsed since vaccination. Any *uniform* rate of increased susceptibility to

small-pox from year to year from the period of vaccination has not been demonstrated; but an increasing susceptibility to small-pox continues up to about thirty years of age at least, after which period of life it seems that the liability to contract small-pox continues to decline (HEIM, MOHL, RETZIUS, MARSON, SIMON).

Dr. Balfour, of the Army Medical Department, adverted some time ago to these important facts; and there can be no doubt of the practical result to which they point—namely, re-vaccination, as a most necessary supplemental measure to vaccination. A large reduction in mortality, and in the occurrence of small-pox, can be shown to have taken place from the practice of re-vaccination, so as to leave no doubt of its practical efficacy. The records of the earliest experience of its usefulness date from Wirtemberg, 1829 to 1836. In 1833 between 40,000 and 50,000 adults were *re-vaccinated* in the Prussian army, and in about 33 *per cent.* of the entire number this *re-vaccination* “took” with perfect success. Amongst Russian soldiers at Kasan, the rate of perfect success was about 18 *per cent.* In the army of Denmark, from 1843 to 1847, nearly 20,000 *re-vaccinations* were practised, of which more than a half were attended with *perfect* success. and more than a quarter with *modified* success. Since 1843 *re-vaccination* has been compulsory in the Bavarian army. From that date till 1857 not even a single case of unmodified small-pox has occurred, nor a single death from small-pox. Similar good results have followed the institution of re-vaccination in the Danish army, the army of Sweden, of Baden, and in the British army also, according to Dr. Balfour’s interesting report for 1859. So great, indeed, is the practical importance of *re-vaccination*, that in the British army a departmental order was issued by circular, of date 21st September, 1858, and is at present in force, which ordains that “every recruit, without exception, on joining the Head-Quarters or Depot of the Corps or Regiment to which he belongs, shall be vaccinated, even if he should be found to have marks of small-pox or of previous vaccination, and that a monthly return of the results (as to (1.) a perfect vaccine pustule following the operation, or (2.) a modified one, or (3.) a failure) shall be forwarded to the Director-General” (*Statistical Report* for 1859, p. 21).

On the other hand, it must be remembered, as Mr. Marson clearly shows, that “probably *re-vaccination* does not afford the

same amount of protection that the first vaccination *well performed* does. The great object to aim at is to vaccinate *well* in infancy. This should be looked upon as the sheet anchor; and therefore a careless vaccination should be deprecated at all times, practised under the belief that, if it fails to take effect properly, it will be of no consequence, as the operation can be repeated. By such a proceeding the vaccination often takes effect *badly*, and will never afterwards take effect *properly*, and the individual may take small-pox *severely*."

It has been alleged (but sufficient proof has not yet been adduced to show) *that the vaccine virus becomes deteriorated by its passage through numerous human bodies*. In other words, it has been supposed that its protective influence is weakened by length of time or of use, in consequence of the long succession of subjects through whom it has been transmitted since its direct inoculation from the cow. This doctrine is opposed to the obvious pathological fact, that the specific virus of cow-pox, small-pox, and other similar diseases, multiplies and reproduces itself in the system of those who suffer in the natural course of these diseases. Considerable differences of theoretical opinion prevail upon the point. In the report of the National Vaccine Establishment for 1854 it is stated "that the vaccine lymph does not lose any of its prophylactic power by a continued transit through successive subjects." Such an unqualified belief is not, however, by any means universal, as shown in various parts of the evidence collected by Mr. Simon. It is certain that the vaccine lymph, when taken direct from the cow, seems to show an amount of infective power which is not usual in lymph of long descent; but how much of this effect is due to irritation simply, and how much to specific action, does not seem certain. Lymph direct from the cow "takes" (as the phrase is) in persons with whom lymph of long descent has failed. This is more often obvious in re-vaccinations. Lymph direct from the cow excites local changes of an intenser kind, so active, indeed, as to render caution necessary in its selection and use. The vesicle produced by it runs a full course, compared with which the progress of vaccine vesicles from lymph of long descent seems unduly rapid, and their termination premature. Also, the lymph direct from the cow renders more certain, and apparently more characteristic, that slight febrile disturbance which is proper to the action of cow-pox on

the human system. This febrile disturbance is undoubtedly an essential pathological phenomenon, demanded alike for the due protection of the vaccinated person and for the perfect development and local multiplication of laudable and efficient lymph at the spot where the specific vaccine virus was originally implanted. The more distinctly and typically the specific febrile action is expressed which follows the implanting of the vaccine virus, the more certainly is the person protected, and the more efficient is the local development of the lymph which has been multiplied at the site of inoculation. The development of any other febrile state, such as from cold, or other disease, is apt to hinder the development and progress of the vaccine vesicle altogether. Referring to the records of re-vaccination in the Prussian army, an extremely interesting fact is brought out by Mr. Simon, tending to confirm the doctrine that, by transmission through a succession of persons, the vaccine virus has degenerated—namely, *“that the re-vaccinations of 1836, as tested by eventual resusceptibility to cow-pox were not half so stable as the vaccinations of 1813.”*

On the whole, therefore, there appears to be still room to believe that any diminution of protective influence from vaccination may be due to personal carelessness—first, in the *selection* of lymph for use, as well direct from the cow as of lymph of long descent; and secondly, in the choice of cases to continue the vaccinations from—cases, for example, being chosen where the lymph of the local vesicle at the site of vaccination has been developed in the absence of the constitutional specific febrile phenomena; and in which the lymphic contents of the vesicle are not only impotent, but the anatomical development and structure of the vesicle in respect of its dissepiments are at the same time incomplete and imperfect. The cicatrix, scar, or mark left by imperfect vaccination is also an imperfect cicatrix, and is capable of recognition as such. To an almost incalculable extent, the *protective power* of vaccination has been impaired by imperfect vaccination, as shown by Mr. Marson—a fact which does not seem to be duly appreciated as yet, either by the Medical Profession or by the Public.

The Operation of Vaccination ought to be performed in childhood, and it is ordained by law in this country to be performed within three, or in case of orphanage, within four months of

birth. The infant ought to be, at least, from *four to six weeks* old, before a disease, sometimes attended with considerable febrile disturbance, is ingrafted upon the constitution. Under *six weeks* of age, infants should never be vaccinated, unless in cases of urgent necessity, such as small-pox being in the vicinity. The age of *three months* is on the whole to be preferred. The child ought to be in good health, free from any eruptive cutaneous disease, and free from disorders of teething, of the bowels, or other diseases peculiar to the age of childhood, otherwise the protective influence of the vaccination cannot be depended on.

Difference of opinion exists as to the number of vesicles it is proper to graft upon the arm, and the size of them. Some believe the person to be as thoroughly protected by a small vesicle, "the tenth of an inch in diameter, as if the arm were covered with inoculated points" (CAZENAVE, ANDREW ANDERSON); and many vaccinators regard the multiplication of vesicles only as a safeguard against failure, and attach value to *one* successful insertion only of the vaccine lymph (Buchanan, *Appendix to Fourth Report on Public Health*, for 1861, p. 111). On the other hand, the official instructions issued to vaccinators in England contain the following directions:—"In all ordinary vaccinations, vaccinate by *four or five separate punctures*, so as to produce *four or five separate good-sized vesicles*; or if you vaccinate otherwise than by separate punctures" (for some vaccinators prefer to make long scratches, side by side or intersectingly, instead of punctures), "take special care to secure the production of *four or five separate good-sized vesicles*." This is considered necessary for securing to those who are vaccinated the full amount of protection which good vaccination confers. The superior value of several vesicles is especially insisted upon by Marson, Seaton, and Simon. They have shown a constant relation to subsist between the *number* of the sufficient cicatrices and the degree of protection afforded.

The skin covering the insertion of the deltoid muscle is the place generally chosen for implanting the specific lymph of variolæ vaccinae. The methods of operating are as follows:—

1. The part of the arm to be operated upon should be grasped with the left hand, and the thumb of that hand should draw the skin with sufficient tightness, so as to facilitate the introduction of the point of a lancet with the other hand. Three or four punctures should be made near each other, for each intended

vesicle. These punctures should penetrate the cuticle to the extent of a few lines in an oblique direction, so as to make a minute valvular aperture, and so as to impinge upon or penetrate the *cutis vera*. The lancet used to make the puncture should be charged with the vaccine *virus*. It should be allowed to remain in the punctures for *several seconds, and, in the course of its removal, the site of puncture should be compressed for a moment or so*, to prevent bleeding, and also to retain the virus from the lancet's point. In the case of several punctures, it is advisable to use "*points*" of ivory or quill, or of the teeth of a comb, charged with the virus. These should, on the withdrawal of the lancet, be inserted into the punctures, and allowed to remain for several seconds, to be removed in the same way as that in which the lancet charged with the virus is removed.

2. Another mode of operation is often chosen—namely, to make an immense number of minute scratches over a very limited area of skin, and as close together as possible. In this way the number of groups of scratches will correspond to the number of vesicles intended to be ingrafted. The scratches may be made with the point of a clean lancet, and may be either parallel to each other or crossed in two or any number of directions. The number of these groups of scratches will vary according as three, four, five, or more vesicles are considered necessary, and the length of the individual scratches will determine the size of the resulting vesicle, and, to some degree, the soreness of the arm. It is necessary to remember these facts in dealing with young and delicate children, so as not to give rise to unnecessary suffering, torment, and danger. The scratches should be so slight as barely to result in the faintest possible exudation of blood, and that only after the lapse of a second or two. To the group of scratches from which blood first exudes, the "*point*" or lancet is to be applied, charged with the specific *virus*. The lymph containing this *virus* will be at once absorbed; and the blood with which the lymph is mixed should be smeared over and pressed into the other scratches in succession several times.

3. Simple abrasion of the cuticle is sometimes resorted to with very good success—namely, by scraping off the cuticle with the lancet, used as an eraser is used to remove blots from paper (*Fourth Report on Public Health*, p. 107).

Signs of Successful Vaccination.—By the end of the second day

Page
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Perfect Vesicle 8th Day areola
Round Scab 14th - 15th
Scab falls 21 - 25th

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SPECIAL PATHOLOGY—COW-POX.

small spots appear elevated over the sites of the punctures, or over the groups of scratches or abrasions; and these, when examined by a simple lens, are seen to be vesicular, and surrounded by a slight redness. This stage continues for three to four days from the date of ingrafting the virus. About the *third*, but rather towards the *fourth* day, the elevation is more perceptible and more red; and by the *fifth* or *sixth* day a distinct vesicle is obvious upon it, of a whitish colour, having a round or oval form, an elevated edge, and a depressed centre. Late on the *seventh*, or early on the *eighth* day, an inflamed ring or areola begins to form round the base of the vesicle, and with it continues to increase during the two following days. This areola is of a circular form, and its diameter extends from one to three inches. On the eighth day the vesicle appears distended with a clear lymph. This is the day of its greatest perfection, and it is the proper period for obtaining the specific virus for continuing vaccination on others. The vesicle is now circular and pearl-coloured; its margin is turgid, firm, shining, and wheel-shaped.

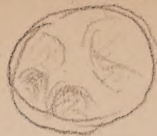
—Having reached its height on the *ninth* or *tenth* day, the development of the bright-red areola is accompanied with considerable tumefaction of the skin, with hardness and swelling of the subjacent areolar tissue. This erythematous ring is often the seat of small vesicles. By the tenth day, also, the febrile symptoms of constitutional disturbance are well expressed, the lymphatics of the arm are engorged, and sometimes a roseolous rash supervenes over the body. On the tenth or eleventh day the areola begins to subside, leaving, as it fades, two or three concentric circles of redness. The vesicle now begins to dry in the centre, and acquires there a brownish colour. The lymph which remains becomes opaque and gradually concretes, desiccation commences, and tumefaction subsides, so that by the fourteenth or fifteenth day the vesicle is converted into a hard round scab of a reddish-brown colour. This scab contracts, dries, and blackens, and about the twenty-first to the twenty-fifth day from the date of vaccination may fall off. It leaves a cicatrix which commonly is permanent in after-life. Indeed, the mark of a good cicatrix is indelible if it is not injured (GREGORY, MARSON, CEELY, CAZENAVE, SIMON).

(4) While these local changes are in active progress, febrile pheno-

mena become established—first, so slightly from the fifth to the seventh day, that often the fact passes unobserved; and again, more considerably during those days when the areola is about its height. The patient is then restless and hot, with more or less disturbance of stomach and bowels. About the same time, especially if the weather be hot, children of full habit not unfrequently show on the extremities, and less copiously on the trunk, a lichenous, roseolar, or vesicular eruption, which commonly continues for about a week. When vaccination is performed on such adults or adolescents as have not previously been vaccinated, and likewise when lymph is employed which has recently been derived from the cow, the resulting phenomena, as compared with the preceding description, are somewhat retarded in their course, and the areola is apt to be much more diffuse. There is also more feverishness, and eruption is less frequently seen (Simon, *Health Report*, 1859).

Signs of Successful Re-vaccination.—When persons who have once been efficiently vaccinated are, some years afterwards, re-vaccinated with effective lymph, there sometimes result vesicles which, as regards their course and that of the attendant areolæ, cannot be distinguished from the perfect results of primary vaccination. But far more usually the results are more or less modified by the influence of such previous vaccination. Often no true vesicles form, but merely papular elevations surrounded by areolæ; and these results, having attained their maximum on or before the fifth day, afterwards quickly decline. Or, if vesicles form, their shape is apt to vary from that of the regular vesicle, and their course to be more rapid, so that their maturity is reached on or before the sixth day, their areolæ decline on or before the eighth day, and their scabbing begins correspondingly early. In either case the areolæ tend to diffuse themselves more widely and less regularly, and with more affection of the areolar membrane, than in primary vaccination; and the local changes are accompanied by much itching, often by some irritation of the axillary glands, and in some cases on the fourth or fifth day by considerable febrile disturbance, (SIMON, l. c.)

Characters of the Cicatrix after Vaccination.—It seems now to have been agreed to arrange the characters of cicatrices after vaccination into the following *three* classes:—



1. "*Typical*," "*excellent*," "*perfect*," "*good*," or "*first-rate*" cicatrices are recognized by their circular form and pale or white appearance. They are somewhat depressed, and dotted, indented, or foveolated with minute pits or depressions over the base, supposed to indicate the number of compartments in the anatomical structure of the vesicle (referred to at pages 236 and 237). In some instances there are radiations from the centre. It has been considered that the normal diameter of a cicatrix produced by a single insertion is one-third of an inch; that scars of larger measurement are generally of double or multiple origin.

2. "*Fair*," "*passable*," or "*modified*" cicatrices possess the characters of the typical cicatrix, but they are less perfectly expressed, the contour being less regular, and the size just *within* the average above mentioned. To irregularity of contour, however, it must be remembered that scars resulting from single insertions (as in the ordinary method of puncture) are notably uniform, so that irregularity of contour, when associated with a single puncture for vaccination, indicates that the progress of the vesicle has been irregular; but where the scar results from several contiguous insertions or scratches, no such inference can be made.

3. "*Bad*" cicatrices, which must be held as denoting "failures," are such scars as cannot be recognized as the product of vaccination, by any circumstance beyond being found near the usual site of the operation. Scars also having a less diameter than a quarter of an inch ought to find a place amongst this class; and generally, all ill-defined, faint, scarcely discernible white patches, especially such as consist of large, flat, ill-defined shiny marks. Fruitless attempts at vaccination may be also recognized by the permanent traces left of the parallel or transverse scratches employed at the operation.

It is, however, very difficult to describe the extent of differences between the results produced by different vaccinators. A large amount of *bad*, and a still larger amount of *second-rate* vaccination has been found to prevail in many districts, as the result of the inspections instituted by Mr. Simon in 1860 and 1861 abundantly testify. Medical men are found to vary exceedingly in their estimate of a satisfactory vaccine vesicle and cicatrix, or

the reverse, for the standard is comparative rather than absolute (SEATON, SANDERSON, BUCHANAN). This is exactly what might have been expected, seeing that medical students are left to pick up their knowledge of vaccination where they can. In fact, practical medical education at our schools of medicine has hitherto, or until very recently, been entirely *nil* in regard to this most important subject; and no test of knowledge has ever been applied. Many men, whose estimate of the quality of the resulting cicatrices is of a low standard, can scarcely appreciate the typical character of marks which are the ordinary results of good vaccination (SEATON). Excessively small cicatrices are apt not to be perfect, and there are great varieties in the size of cicatrices of perfect character, the results of puncture. It is therefore fairly presumed that cicatrices which thus vary cannot all have precisely the same value. The hand of different vaccinators can even be recognized by the kind of marks they leave behind them. The marks of some vaccinators are conspicuous for their excellence; the marks left by others are not so; and hence there are great differences between the vaccination of districts where different vaccinators are employed. In the schools, for instance, of large towns, Mr. Seaton informs us that "where the work of many vaccinators was seen together, it was frequently possible to fit the work to the vaccinator by the kind of cicatrix."

With regard to the means of estimating the efficiency of vaccination, it seems established that "*a distinct connection subsists between the NUMBER and the QUALITY of the cicatrices and the protection conferred by vaccination against small-pox; so that it may be confidently stated that that vaccination is the most efficient from which the most and the best cicatrices result.*" The evidence derived from the records of the Small-Pox Hospital, collected by Mr. Marson, regarding the superior value of several rather than few vesicles, appears to be conclusive on this point.

These facts have been tabulated by Mr. Simon in the following form, as the result of observations made during twenty-five years, in nearly 6,000 cases of small-pox contracted after vaccination, the persons having been vaccinated in different ways as regards the *number and quality* of the cicatrices:—

Cases of Small-Pox Classified according to the Vaccination Marks or Cicatrices borne by each Patient respectively.	Number of DEATHS <i>per cent.</i> in each Class respectively.
CLASS I. Stated to have been vaccinated, but having } NO cicatrix, ... }	21 $\frac{3}{4}$
„ II. Having ONE vaccine cicatrix,*	7 $\frac{1}{2}$
„ III. Having TWO vaccine cicatrices,†	4 $\frac{1}{8}$
„ IV. Having THREE vaccine cicatrices, ...	1 $\frac{3}{4}$
„ V. Having FOUR or MORE vaccine cicatrices, ...	$\frac{3}{4}$
UNVACCINATED, ...	35 $\frac{1}{2}$

Looking, therefore, to the characters or quality of the cicatrices, and to the number of the vesicles which have given rise to these cicatrices, FOUR DEGREES of protection conferred by vaccination may be specified, and the community inspected may be arranged into the following FOUR classes:—

CLASS I. Best protected—having more than two TYPICAL marks.

CLASS II. Sufficiently well protected—having two TYPICAL marks.

CLASS III. Moderately protected—having two or more PASSABLE, or one TYPICAL mark.

CLASS IV. Badly protected—having BAD marks, or having only one PASSABLE mark.

B Selection of Lymph for Vaccination.—The lymph used for vaccination ought to be taken from the vesicle on the EIGHTH day—the day-week after the operation—when the lymph is yet clear and the vesicle turgid, firm, shining, pearl-coloured, and translucent, and before the vascular zone has reached its full development. The lymph ought not to be taken from any but perfectly “typical” vesicles. Inferior, or merely passable vesicles, ought not to be used to propagate lymph. Small vesicles, exhausted vesicles, or vesicles far advanced (such as tenth or twelfth day) are to be avoided. Very early lymph appears, as a rule, to give

* Among cases in which the one cicatrix was *well marked* or *typical* the death-rate was 4 $\frac{1}{4}$. Among cases in which it was *badly* marked the death-rate was 12.

† Among cases in which the two cicatrices were *well marked* the death-rate was 2 $\frac{3}{4}$. Among cases in which they were *badly* marked it was 7 $\frac{1}{4}$.

the worst cicatrices. Thus "the careful vaccinator does not indifferently vaccinate from the arms of all infants brought back on the *eighth* day, but exercises selection among them. The fresh lymph from the vesicle ought to be ingrafted *directly* upon the arm of the child about to be vaccinated—"arm-to-arm" vaccination, as it is called. Dry, or otherwise preserved lymph ought only to be used when fresh lymph cannot possibly be obtained. Properly dried lymph, however, seems capable of producing quite as good results as arm-to-arm vaccination; but it demands incomparably more care than it generally receives, first in its storage and afterwards in its use. It may be dried and stored on "points" of ivory or bone, or upon small pieces of glass glued together by the dried lymph, or on lancets. These should be well charged—*i. e.*, coated twice, or even thrice with the lymph, and rolled up in a covering of goldbeater's skin, and still further secured from atmospheric influences by an outer case of tinfoil hermetically sealed, or in a phial carefully corked, in which they may be packed with cotton, if they require to be transmitted to any distance. Glycerine has been used with success to keep the lymph liquid.

By proper care, complete and perfect vaccination may be attained under every variety of method; but bad vaccination, as it prevails at present, is almost always directly dependent on the careless employment of improperly preserved *dry* lymph, and indirectly associated with irregularity of inspection, in consequence of which the vaccinator remains unaware of the number and extent of his *failures*, and loses all the advantages of experience. "The use of the capillary tubes of Dr. Husband affords considerable advantages to the public vaccinator, especially if his district be rural—*Firstly*, Because it furnishes him with an efficient means of maintaining his supply without having recourse to extraneous sources, and thus enables him to dispense altogether with the use of 'points,' 'glasses,' &c.; *Secondly*, Because in thinly populated neighbourhoods, in which experience shows that it is impossible to assemble all the children at any particular station, it enables him with equal advantage to vaccinate from house to house" (Sanderson in *Public Health Report*, 1861). For a detailed account of Dr. Husband's method of preserving lymph, the reader is referred to the *Second Report of the Medical Officer of the Privy Council*, 1859.

CHICKEN-POX.

LATIN, *Varicella*; FRENCH, *Varicelle*; GERMAN, *Windpocken*—Syn., *Wasserpocken*, *Varicellen*; ITALIAN, *Varicella*.

Definition.—*The disease consists of a specific eruption, in a series of crops, on the breast, back, face, and extremities, attended with fever, which runs a definite course in eight or ten days.*

Pathology.—This disease derives an importance which it does not of itself possess, in consequence of its resemblance to small-pox, with the modified form of which it is considered by some to be identical. It is for the most part peculiar to childhood and early adult age; but its epidemic influence is very inconsiderable, and its extension easily under control. That it is communicable has been proved by inoculation. The theory of the disease, therefore, is, that a specific poison, after a given period of latency, gives rise to primary fever, which lasts from twenty-four to seventy-two hours, when the eruption appears, and runs a course of eight or ten days. The fever is much mitigated on the appearance of the eruption, and entirely subsides with it.

That fever precedes the eruption is a phenomenon observed so generally that no exception is to be found in the account given of the disease by any writer excepting Heberden. The febrile affection is of a mild character, and though for a few hours it may seem severe, yet, perhaps, it never passes into a stage so severe as to have the tongue of a brown and coated appearance. The eruption has three stages,—that of pimple, of vesicle, and of incrustation; and after the fever has lasted from twenty-four to seventy-two hours, a number of red papulæ appear, which become vesicular, and perhaps in a few points pustular, on the first day. On the second day the vesicles are filled with a whitish or straw-coloured lymph. On the third and fourth days they attain their greatest magnitude, when they become *acuminated*, and shortly afterwards they burst and shrivel, except those which contain purulent matter, and have much inflammation around their base. The fifth day they begin to crust, and in four or five days more the crust falls off, leaving for a time red spots on the skin, generally without, but sometimes with a “pit” or depression. The “pit” is permanent, and the cicatrix generally whiter than the original tissue, and the patient consequently is marked or scarred. The eruption is not at first universal over the body, but

usually consists of a series of crops, which succeed each other at intervals of twenty-four hours, and die away in the order of their occurrence. The first crop usually appears on the breast and back, and afterwards on the face and extremities. The number of crops may be limited to two or three, while in other cases a new succession will appear every twenty-four hours for ten or twelve days.

Symptoms.—Of the varicella there are three forms, the *varicella lenticularis*, the *varicella conoides*, and the *varicella globata*. The symptoms of these varieties are similar to each other, their only differences consisting in the size and form of the vesicle—that of the *varicella globata* being the largest.

The fever which precedes this eruption is often as severe as that which precedes mild small-pox or measles; but it generally, though not constantly, remits on the appearance of the eruption, and does not return as the eruption approaches maturity. The urine, however, is usually little affected in the early stages, when it is often as limpid as in hysteria; but when the fever runs high, it assumes the usual febrile characters (PARKES).

The globate chicken-pox is known as the *swine-pox*, or, vulgarly, “*the hives*.” The eruption consists of large vesicles not quite circular in form, but often a little larger than the pustules of small-pox, surrounded with a red margin, and containing a transparent fluid, which, on the second day of the eruption, resembles milk whey. On the third day they subside, shrivel, and present a yellow tint. Before the conclusion of the fourth day they are converted into thin blackish scabs, which dry, and fall off in four or five days more.

Diagnosis.—Dr. John Thomson, who carefully studied this disease during the epidemics of 1815 to 1821, concluded that it was impossible to distinguish chicken-pox from modified small-pox; and as their identity is still a matter of opinion, the following statements (CRAIGIE) embrace the most important pathognomonic characters derived from the respective phenomena of both diseases:—

1. Chicken-pox emits a peculiar odour, different from that of small-pox, and less decidedly partaking of the variolous fetor.
2. Chicken-pox appears indiscriminately, and almost equally all over the person, beginning first on the trunk in general, and then appearing on the face and scalp; while small-pox appears first on the face and neck, and the pimples are more numerous on the face than on any other part.

3. Chicken-pox eruption is generally completed in the space of twenty-four hours, or solitary vesicles come out irregularly afterwards in different points; but in small-pox the eruption begins in the evening of the third, or morning of the fourth day, and proceeds regularly for the ensuing three days, until it is completely established.

4. While variolous pustules are on the first and second days of the eruption small, hard, globular, red, and painful, and communicate to the finger a sensation similar to that which would be excited by the presence of small round seeds under the cuticle;—in chicken-pox, every vesicle almost has on the first day a hard red margin, but communicates to the finger a sensation like that from a rounded seed flattened by pressure.

5. On the second or third day of the eruption of chicken-pox, the individual bodies are vesicles containing serous fluid, and giving them a whitish aspect.

6. These vesicles are surrounded by little or no inflammatory redness, and do not naturally, and independent of external violence, proceed to suppuration.

7. Chicken-pox may be confidently distinguished from small-pox on the third and fourth days by the state of the vesicles, some of which, being left entire, are shrivelled and wrinkled, while others, whose ruptured tops have been closed by incrustation of their fluid, are marked by radiating furrows. None present depressions on the *apices*; and as they do not suppurate, they incrust and disappear sooner than variolous pustules.

8. The marks left by chicken-pox, when they do leave marks, present a peculiar conformation, being round or elliptical, and less frequently irregular than those of small-pox, and in general smooth and shining. Lastly, it is said by Luders, that while small-pox is formed in the *cutis vera* or corion, the chicken-pox eruption is formed in the tissue situate between the corion and cuticle (CRAIGIE, vol. i., p. 614).

Treatment.—It consists simply in abstinence from animal food, having recourse to a milk diet, and careful attention to the bowels. The patient is to be kept cool, by light coverings, and by making him repose on a mattress rather than on a feather bed.

MILIARY FEVER.

LATIN, FRENCH, GERMAN, AND ITALIAN, *Miliaria*.

Definition.—*A disease in which there is an eruption of innumerable minute pimples, with white summits, occurring in successive crops upon the skin of the trunk and extremities, preceded and accompanied with fever, anxietas, oppression of*

respiration, copious sweats of a rank, sour, fetid odour, peculiar to the disease. The base of the pimples and the skin around are red and irritable.

Pathology.—As to the specific nature of this disease pathologists are not agreed. All physicians are not disposed to admit that in *miliaria* a peculiar specific disease exists, with a characteristic eruption and definite course, such as the variolous pustules and course of small-pox exhibit; by many it is believed that “this affection is almost invariably symptomatic.” Certain it is, however, that a peculiar epidemic disease prevailed in different parts of Europe at different periods in the world’s history, the nature of which is described in the definition; and although in this country it seems to have disappeared, yet there can be no doubt that a specific disease of this description prevails epidemically in many parts of continental Europe and Asia. The disease of these epidemics has been described under the various names of “sweating sickness,” “miliary fever,” “sudatoria,” “miliaria,” and the like. Rayer has given the most accurate account of the disease; and I had an opportunity of witnessing a great number of cases of it amongst the Turks, in their military hospitals at Scutari, during the war against Russia in 1854-1856. The temperature and physical climate of that place, combined with the relaxed habits of the Turks, appear to be favourable to the development of such a disease. The best accounts of it are those of Borsieri and Rayer.

Symptoms.—The fever which precedes the eruption is ushered in by chills, intense and general, shivering, anxietas, oppression of the chest, restlessness, a sense of great feebleness and imminent fainting, with pains in the head, loins, and limbs. In a few hours, nausea, flushing, and profuse sweating supervene, but without any diminution of the dyspnoea, the anxietas, or pectoral oppression, but rather with an aggravation, in the form of short, irregular, panting, and sighing breathing, as if proceeding from a sense of weight under the sternum, with a feeling of internal heat, wandering pains, and sometimes cramps of the hands and calves of the legs. The pulse is generally rapid, small, and feeble; in a few cases hard; often variable, irregular, or intermittent at every ninth, twelfth, or sixteenth beat. The tongue is coated with a white, foul, or yellow fur, indicative of a sluggish condition of the alimentary canal; and the bowels are constipated throughout the disease. The sweat which accompanies this febrile state is

profuse, and emits a peculiar smell of a rank, sour, fetid odour. From the fifth or sixth day, up to the twenty-first, an itching sensation is felt in the mammary and epigastric regions, and inner surface of the arms, and the skin of those parts is found to be diffusely red, rough, and irregular, with numerous elevations not larger than pin-heads. In a short time the summits of these become pearly-white, the cuticle being elevated by a slight opaque sero-albuminous fluid,—crop after crop breaks out, and continues from three to seven days, followed by a corresponding desquamation of the cuticle. This eruption is generally confined to the neck, breast, mammary and epigastric regions, and the inner surface of the loins and legs. In severe cases, miliary vesicles appear at the junctions of the skin and mucous membranes, and there they are apt to become aphthous.

A deranged state of the gastro-enteric mucous membrane, indicated by nausea and vomiting of bilious matter, acid eructations, flatulence, and diarrhoea, frequently complicate the disease. Two forms have been described,—namely, a mild and malignant. The malignant is rendered so chiefly by the occurrence of violent inflammation in some of the internal organs, especially of the stomach, lungs, kidneys, or brain; and the danger of the disease is chiefly due to these complications. Such malignant forms have been known to prove fatal in two or three days, but more frequently in from seven to twenty-one.

The Treatment of the disease appears to consist in cooling drinks, purgatives, and antiphlogistics, as prescribed by the Italian medical officers who commonly attend on the sick in Turkey.

MEASLES.

LATIN, *Morbili*; FRENCH, *Rougeole*; GERMAN, *Masern*; ITALIAN, *Rosolia*.

Definition.—*The eruption in crops of a crimson rash, consisting of slightly elevated minute dots, disposed in irregular circular forms, or crescents; preceded by catarrhal symptoms for about four days, and accompanied with fever. It affects the system only once; and sometimes prevails as an epidemic. The eruption lasts six or seven days, and the whole duration of the disease is completed in from nine to twelve days.*

Pathology.—That a poison is absorbed in cases of measles, and infects the blood, there can be no doubt, inducing, after a period

of incubation of thirteen or fourteen days, a continued fever, which does not remit on the appearance of the eruption. The fever thus established at the end of three, more generally of four, and in some few instances of five days, is followed by a certain secondary or specific inflammation of the skin and of the mucous membranes of the eyes, nose, mouth, fauces, and bronchia. In a few cases the poison has certain tertiary actions, and produces inflammation of the substance of the lungs, or of the pleura, which may greatly prolong the illness.

The pyrexia may greatly vary in intensity, but it is uniformly present. The fever which precedes the local lesions is termed the primary fever; and the premonitory phenomena of cough, sneezing, and general *malaise*, are usually more prolonged than in the other eruptive fevers. It does not always happen, however, that the functions of the mucous membrane are disordered, as well as the cutaneous surface. There are cases in which no catarrhal symptoms exist, and such cases are described as "*morbilli sine catarrho*." Such cases occur during epidemics of the disease, and are but few in number.

Since the affection of the skin is uniformly present, while that of the mucous membranes is sometimes absent, the cutaneous eruption is necessarily the great characteristic of the disease; but the morbillous eruption being evanescent after death, we can only imperfectly trace its pathology. It first appears as a circular spot or blotch, similar to a flea-bite, slightly prominent, and scarcely sensible to the touch. Its colour is of a pinkish red, or deep raspberry hue, and in rare instances, as in the *morbilli nigri*, is livid or black. In severe cases, especially if the patient be of tender age, the eruption assumes a papular form, and, when at its height, occasionally a vesicular form, the latter being most common on the arms, the neck, or the breast. The colour of the eruption is evanescent on pressure, but returns on the finger being removed.

The patches of the eruption are extremely numerous, so that little of the healthy skin intervenes between them; and they not unfrequently become confluent, forming large maculæ, sometimes of a semi-lunar form. The principal seats of the eruption are the face, back, and loins; the parts least affected are the pudendal and popliteal regions. The inflammation of the cutaneous texture extends in some degree to the subjacent

Measles of fever { 5th day perhaps (109)
1st day of eruption

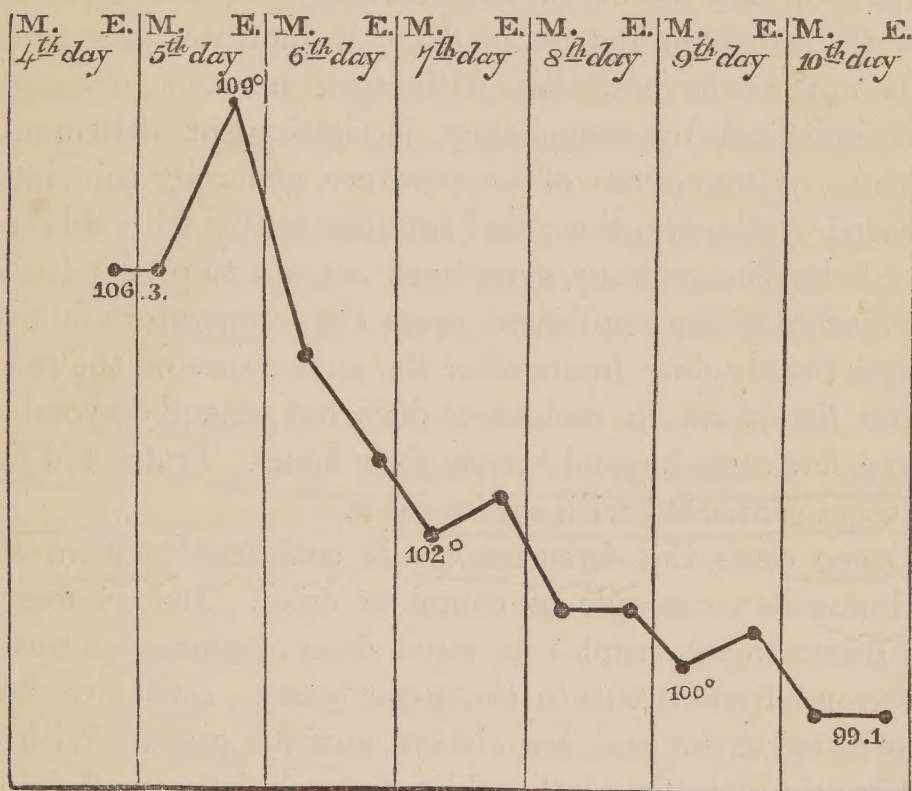
areolar tissue, for the face is tumid and swollen, but not so as to close the eyelids.

The eruption does not at once cover the whole body, but occurs in three crops, each of which follows the other at an interval of twenty-four hours, the duration of each crop being from three to four days. The course of measles, then, in its most simple uncomplicated form, is that on the third or fourth day of the primary fever, which is continuous, the first crop of the eruption appears on the face, neck, and upper extremities; on the following day the second crop covers the trunk; and on the third day the third crop appears on the lower extremities, so that the whole body is covered with the eruption, which is then at its height. On the following day (the fourth of the eruption) it begins to decline from the face, neck, and upper extremities; and on the next day it fades from the trunk. On the sixth or seventh day it is evanescent over the whole body, and terminates by resolution, followed by a furfuraceous desquamation of the cuticle generally. The maximum of the fever is reached about the fifth day, and may last from twelve to twenty-four hours; and it is immediately followed by a rapid and almost complete defervescence, the temperature sinking in one night two or more degrees, (Fahr.) It continues to decrease throughout the following morning and day; and on the second day from the beginning of the defervescence the normal temperature is arrived at. It is only in very severe cases that this steady decrease is prolonged beyond twenty-four or forty-eight hours more. In severe cases the decrease of temperature may be slower and more protracted; but if the defervescence be more prolonged, it is a fair ground for suspecting some untoward complication.

A similar course of temperature, as regards defervescence, has been observed to obtain in cases of erysipelas of the face, but the fastigium lasts longer, and the epoch for the commencement of the defervescence vacillates between the fourth and eighth days.

It is the amount of fever present—*i. e.*, the increased bodily heat, as measured by the thermometer—which greatly aids in deciding diagnosis and prognosis. From this point of view slight, severe, and complicated cases are to be distinguished.

THE FOLLOWING DIAGRAM REPRESENTS THE TYPICAL RANGE OF TEMPERATURE IN A CASE OF MEASLES. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE FOURTH DAY OF THE DISEASE:—



LINE OF NORMAL TEMPERATURE 98° FAHR.

During the period of incubation, slight feverishness, depression, and catarrh are present in adults; but when the disease is expressed, its commencement may be marked by violent shivering, or merely by chilliness, characteristic of catarrhal fever. The inflammation of the mucous membrane, of the eyes, and nasal fossæ, indicated by more or less constant sneezing, generally commences either with or before the primary fever, and consequently precedes the eruption by some days. This inflammation is perhaps, for a few hours, confined to fixed spots, and is marked by itching at the mucous orifices; then it becomes diffuse, and quickly changes to the serous; for a profuse watery discharge from the eyes and nostrils shortly follows, technically termed "coryza." This affection usually continues till the decline of the eruption, and in some cases to a later period. Children, as a rule, are not anxious to

seek their beds; even with a temperature of 104° Fahr. they are still able to remain up; but in cases of pneumonia, with the same temperature, they desire to lie down at once.

The temperature rises rapidly towards the breaking out of the eruption. Its rise is steady to the *fastigium*, but if remissions are marked, they occur in the morning. If the fever is high before the eruption for many days, it indicates a severe case, and is apt to be attended with such nervous derangements as are indicated by somnolency, jactitation, or delirium. The *fastigium*, or maximum of temperature, generally coincides with the period of the eruption; and simultaneously with this increase of the fever the nervous symptoms are apt to predominate. In the majority of uncomplicated cases the temperature falls within the first *twenty-four* hours after the appearance of the red spots, and the *fastigium* in such cases does not extend beyond *twelve*, and in a few cases beyond *twenty-four* hours. Protracted *fastigia* are always connected with severe cases.

In some cases the *defervescence* is completed within *twenty-four* hours—an example of complete crisis. But pauses sometimes occur to interrupt this rapid *defervescence*—pauses which may extend from *twelve* to *thirty-six* hours. Such are examples of protracted crisis, and are always anxious cases. With regard to prognosis, it is found that high fever, having small daily fluctuations, especially with the co-existence of nervous derangements, delirium, and the like, are very unfavourable symptoms. Prolific eruptions are always more favourable than scanty exanthema. Short duration of the *fastigium*, rapid *defervescence*, and speedy disappearance of the eruption, are the most favourable events. On the other hand, exacerbations are always more or less unfavourable.

Of the complications of measles, the most important is catarrhal pneumonia; and a fatal issue is apt to ensue in cases of young children. In such cases the pulse and respirations are enormously accelerated, the face is flushed, and the movements of the body are lively. The contractions of the heart are then apt to abate in frequency, and the breathing to become very inefficient; while simultaneously with diminishing expectoration the breathing surface of the lungs becomes less and less, owing to the bronchia being rendered more and more impervious. Portions of lungs then collapse; and others become emphysematous.

Catarrhal Pneumonia most frequent
Complication
Pleurisy also

The mucous membrane of the mouth and fauces in most of the severe cases inflames, but the inflammation differs from that of the eyes and nose in not being accompanied by any discharge. In other respects it is exactly similar to the cutaneous eruption, for a number of exanthematous patches, more or less confluent, are seen upon the palate, uvula, tonsils, and *velum pendulum palati*, and they, equally, terminate by resolution. They appear also at the same time with the eruption on the face, neck, and upper extremities, but do not decline till the eruption fades from the body generally.

The cough and expectoration, which indicate the attack and accompany it, are constant, and the latter shows that it partakes of the same serous character as that of the nasal and ocular membrane.

When the substance of the lungs is thus affected, a serous exudation pervades that tissue, and the quantity of fluid effused is frequently so considerable as to stream from the lung after death, as soon as its tissue is divided. In severe forms of the disease either the red or grey hepatization of the lung may supervene; but these results are rare. The pleura does not at all times escape the morbid action; and the diffuse, the serous, the adhesive, and even the purulent inflammation, may invade that tissue, and either destroy the patient or retard his convalescence. Few analyses of the urine in cases of measles have been made. Albumen is extremely common in some epidemics, and appears simultaneously with the eruption: it may then disappear, and re-appear during the fading of the rash. Blood in small quantities is also common. In the Leith epidemic of 1854 the recoveries were most speedy when the albuminuria was the greatest (Parkes *On the Urine*, p. 262).

Symptoms.—The symptoms of measles result from the fever and the consecutive local lesions. The varieties of the disease, however, are extremely few, for no instance is known of a morbillous fever without the secondary or specific actions following; but the poison is supposed sometimes to limit its action to one membrane, as the cutis, and to exhaust itself on that tissue; and hence the "*morbilli sine catarrho*." The varying intensity also of the disease has led physicians to consider the phenomena of measles under two grades—namely, the "*morbilli mitiores*" and the "*morbilli graviores*."

The primary fever may make its attack suddenly, or be preceded for a few days with symptoms of a common cold, and in general the latter is the case; but in no instance is the primary fever (which is afterwards prolonged, and accompanies the eruption), at any time, of great intensity. Although many children may die from the severity of the local lesions, yet no instance is known of the patient being overwhelmed or destroyed by the general depressing action of the poison, as is the case in typhus fever or scarlatina. The depressing powers of the poison, however, are considerable, and are often sufficient to confine the patient to his bed for a few days, and to leave him, for a short time after the disease has subsided, weak and debilitated. The type of the fever of measles consequently greatly differs from that of typhus or of scarlatina, and the formidable brown tongue, so grave a symptom in the latter, is hardly known in the former, or only seen in a few fatal cases.

Morbilli Mitiores.

The essential characters of this affection are, that the poison produces primary fever, and a specific inflammation of the skin and mucous membranes,—the defervescence of the fever taking place while the eruption fades.

The symptoms may be divided into three stages: the first embraces the primary fever, or the period before the eruption, and may last from three to five days; while the second stage embraces the period of the eruption, and lasts from six to seven days. These two stages very commonly comprise the whole disease, whose usual course is then from nine to twelve days. The third stage includes any inflammatory action which may be caused by the tertiary action of the poison, and only occasionally exists.

The early symptoms of the primary fever are seldom severe, and greatly resemble those of an ordinary acute catarrh. They are, shivering, alternated with heat, frequent pulse, headache, derangement of the bowels, sometimes accompanied by nausea and vomiting; and these affections are so considerable that the patient usually takes to bed. At the end of a few hours the fever becomes continued, and the specific action of the poison commences by the mucous membrane of the eyes and nose inflaming, so that the light is painful; the senses of smell and

First Stage 3 - 6 days
Second 4 - 5
Third complications

taste are lost, followed by a copious discharge of serum from the nose and eyes, attended with more or less constant sneezing.

The buccal and bronchial membranes may become affected at the same time, and the patient is then troubled with a frequent cough, which has this peculiarity, that it occurs in paroxysms. The cough does not remit till about the seventh day, and is often accompanied by hoarseness, by a sense of constriction across the chest, by diarrhoea, and sometimes by ischuria. The duration of this first stage may be three, four, five, or even six days.

The second stage commences with the appearance of the eruption, whose course and character has been described. On the appearance of the eruption the fever is often aggravated, but the distressing nausea and vomiting seldom last beyond the fourth day. The fever, therefore, together with the coryza, sneezing, coughing, hoarseness, and diarrhoea, continue with unabated severity till the eruption has reached its height, and is fully out over the whole body, which is on the third or fourth day after its first appearance. From this period, in favourable cases, all the symptoms begin to decline; and on the eruption disappearing, the cuticle desquamates, and the disease terminates on the ninth, tenth, or eleventh day from its commencement.

In a few cases, however, on the subsiding of the eruption, or about the ninth, tenth, or eleventh day of the disease, and in some instances earlier, the pectoral symptoms do not subside as they ought to do, but the tertiary actions of the poison are set up, and inflammation of the substance of the lungs or of the pleura takes place, prolonging the duration of the disorder, and endangering the life of the patient. The inflammation of the bronchial membrane is denoted by the expectoration either of a thick viscid mucus or of pus, and which may or may not be streaked with blood; while the mucous or sonorous rattle will point out the peculiar seat and extent of the mischief. If the substance of the lungs be inflamed, the breathing is more difficult, the cough more troublesome, and the countenance livid; but the loud mucous rattle which accompanies it seldom allows us to hear crepitation, or to determine the absence of respiration in any given portion of the lung. If the pleura be inflamed, we have, in addition to the cough, severe pain in the side, and an impossibility of filling the chest with air, except in a very limited degree. This condition is often accompanied by dulness on per-

cussion, by bronchophony or ægophony, assuring us that fluid is effused into the cavity of the chest.

Morbilli Gravioros.

The main characteristic of this severe form of measles is the eruption becoming suddenly black, or of a dark purple with a mixture of yellow. The early writers on measles describe this form of the disease as being much more common in their time than we find it to be in the present day. Sydenham considers this appearance as extremely formidable, and that persons so seized are irrecoverably lost, unless they are immediately relieved by bleeding and a cooler regimen. Willan writes that he has seen this discoloration, but thinks more lightly of it.

The eruption is sometimes greatly delayed from causes not quite manifest. Excessive purging is thought to have this effect, or anything which greatly debilitates the system, hereditary or acquired unhealthiness of constitution, or the peculiarly malignant nature of the disease. The occurrence of the eruption is therefore to be looked for with anxious care, as the appearance of it, even though late, is in itself a favourable indication.

X If the eruption suddenly disappears, or "goes in," it is no less an unfavourable omen, and is apt to be followed by dangerous results, diarrhœa, dyspnœa, coma, convulsions, all which unfavourable signs may again disappear on the re-appearance of the eruption.

MB (**Diagnosis.**—The diseases with which measles may be confounded are scarlet fever and some forms of syphilitic eruptions. The diagnostic symptoms between measles and scarlet fever are numerous; for there are many differences, both in the general course of the fever, the ranges of temperature, and particular symptoms of these diseases, by which they may readily be distinguished from each other. Thus, the periods of the latency of the poisons are different—that of scarlet fever being from two to ten days, while that of measles is from ten to sixteen days. The eruption in scarlet fever seldom appears later than the second day of the primary fever; in measles it is delayed till the fourth day. In scarlatina the exanthematous patches are large, and the surface they cover ample; but in measles they are not larger than flea-bites, and when most confluent the clusters are small, sometimes forming crescentic patches. The colour is also dif-

ferent, being of a bright red in scarlet fever, while in measles it partakes more of a pinkish red or raspberry hue. The affections of the mucous membranes are also different in the two diseases. In scarlatina the tonsils are almost always greatly enlarged and ulcerated, while in measles they are little or not at all affected. In scarlatina the eyes are free from coryza, while in measles this is the most prominent symptom. The tertiary actions of the poison are also different, being, in scarlatina, inflammatory affections of the joints, and dropsy; while in measles they are inflammations of the lungs or pleura; and, lastly, in measles the fever usually subsides on the disappearance of the eruption; but in scarlatina the fever often continues many days or weeks after the eruption has run its course, or till the sore throat has healed.

Prognosis.—The mortality from measles greatly varies in different years. During each of the four years previous to 1858 the proportion of deaths from measles in every 1,000 deaths from other causes has been, in 1851, 24·107; 1852, 14·599; 1853, 11·818; 1854, 21·463. Percival says, that out of 3,807 cases of measles, 91 died, or 1 in 40. Watson says, that in one year, at the London Foundling Hospital, 1 in 10 died; and in another 1 in 3. In the same establishment in 1794, out of 28 cases none died; in 1793, out of 69 cases, 6 died; in 1800, out of 66, 4 died: and the aggregate of these data will give us an average of 1 death in 15: so that the prognosis in every case of measles is favourable in the first instance. The prognosis, however, is more favourable in the country than in large metropolitan towns; for it appears by the Registrar-General's reports that the proportion *per cent.* of the population that died of measles in London is much greater than in England and Wales.

The chief danger arises from bronchial and pulmonary inflammation, and the danger of this is greater after the disease has begun to decline than during its progress. An epidemic of measles occurred at Kiel in 1860. In the fatal cases the chief cause of death was a peculiar state of the lungs, which in part were collapsed, with *foci* of purulent infiltration in various parts, or a condition of carnification. Intense bronchial catarrh was present, extending to the minuter ramifications of the air-tubes, but not of a croupal character (Virchow's *Arch.*, vol. xxi., p. 65; *New Syden. Society Year-Book*, 1861, p. 132). In strumous patients measles may end in the development of miliary tubercles

in the lungs; increasing cough, emaciation, and a harsh, dry skin being the symptoms of such an untoward result.

Croup sometimes supervenes, and cuts off young patients. It tends to be of the asthenic type, and is not unfrequently preceded by diphtheritic inflammation of the fauces, which gradually passes down to the larynx.

Diarrhoea is another danger to be encountered. During convalescence there is a tendency to looseness of the bowels, but which, if moderate, ought not to be counteracted, as it is commonly rather advantageous; but if suffered to continue, the consequences may be fatal.

Catarrhal ophthalmia, if the constitution be strumous, must also be watched for, and, if possible, prevented.

Measles, in any of the malignant forms described, is highly dangerous; and the danger is greater in the old than in the young—in cold than in warm weather.

Causes.—Measles were first noticed at the same time and in the same country with scarlet fever, and the two diseases have subsequently followed nearly the same course. They now prevail all over the world, are little influenced by season, are believed to be constantly in existence somewhere, and occasionally epidemic.

Measles, though incidental to every period of life, are most frequently contracted in childhood, when it is difficult to trace the effects of accidental circumstances, so that our knowledge of the predisposing causes is most imperfect. Both sexes, however, appear to be equally liable to this affection. With respect to the influence of season, it is generally supposed that measles break out most readily in the beginning of winter, increase till the vernal equinox, and then tend to subside towards the summer solstice. The deaths, however, from this disease, registered in England and Wales, show that the influence of season is exceedingly trifling.

Propagation of the Disease by Direct Communication and Infection.

—It is admitted by all authors that a patient labouring under measles generates a poison which may be communicated directly, or which may contaminate the atmosphere with an impalpable poison. Like scarlatina, measles is thus eminently communicable; and in like manner no susceptible person can remain in the same room, or even in the same house, with an infected person, without

hazard of taking the disease. In the year 1824 it was imported into Malta by some children belonging to the 95th regiment, and spread extensively in that island, so that many natives died. This circumstance was the more remarkable, as measles had not been in the island for many years. The *infecting distance* of this poison, it will be plain from what has been stated, must be considerable; indeed, it is often very difficult to isolate the disease in public schools, or other large establishments, where it sometimes appears.

The fact of measles being communicable has often been proved; but some difference of opinion exists as to the possibility of communicating the disease by inoculation. Healthy children have been inoculated, either by blood drawn from the arm of a patient suffering from measles, or with serum taken from the vesicles which are occasionally found intermixed with the eruption,—an experiment which appears to have been first made by Dr. Home, with a view of producing a mild disease; but as no such result has been obtained, the practice has been abandoned. Many trials of this kind have failed to produce the disease, yet, on the whole, successes are sufficiently numerous and varied to warrant the statement that a specific poison communicates the disease.

This disease is also propagated by *fomites*. The strictest demonstration of this fact is, that the disease has been communicated by direct application of substances impregnated with the virus in the attempts to inoculate the disease; it is also proved by the fact that children's clothes, sent home in boxes from schools where the disease has raged, communicate the disease; and also by the same circumstance resulting when susceptible children have lain in the same beds, or in the same room, shortly after it has been occupied by patients suffering from the disease. Cold weather appears favourable to the development and propagation of measles. No age is exempt, from the foetus in the womb to the second childhood of old age; but it is much more frequent in children than in adults, and there are few who have not an attack of measles at some period of life.

The morbillous poison having once produced its specific effects, as a general principle, leaves the patient exempt from all liability to a second attack. This law may be considered as proved both by Willan and Rosenstein—the former affirming that, after an attention of more than twenty years to eruptive complaints, he had not

Latency 10-16 days

met with an individual who had twice had "febrile rubeola;" while the latter states that in a practice of forty-four years he had met with no instance of a second infection. There are, however, occasional exceptions to the rule. One variety of this disease—namely, the *rubeola sine catarrho*—is supposed to afford no protection against an attack of the *rubeola vulgaris*. There are many exceptions, however, to the non-susceptibility of persons who have passed through the *rubeola vulgaris*; for Burserius, Robedieu, Home, Baillie, Rayer, and Holland, have all seen instances of a second attack of the measles in the same individual.

The period of latency of the poison of measles is determined to vary from ten to sixteen days. It seems also ascertained that the specific poison of measles is generated as soon as the primary fever is established, and before the eruption appears.

Treatment.—The nature and course of measles differ from scarlet fever not only in the fever being much less depressing, but in running a shorter and more certain course, and in having no tendency to terminate in ulcerations or mortifications of the skin. The constitution during measles is little impaired by the short continuance of the disease, and consequently admits of a more strictly antiphlogistic treatment.

As no antidote is known to the poison of the measles, the disease must run its course. The rule, therefore, is to interfere as little as possible as long as the disease is pursuing its normal course, and merely to attempt to moderate and subdue symptoms when they threaten danger.

(1) The *morbilli sine catarrho* is usually of such a mild form as to require no other treatment than a milk diet, the customary attention to the bowels, and the prevention of exposure to cold and wet. Measles will not bear exposure of the surface of the body to cold so well as either scarlatina or small-pox, on account of the great tendency to bronchial and pulmonary inflammation. Children must therefore be watched night and day to prevent them lying uncovered, and special care must be taken to avoid exposure to cold during convalescence. In the *morbilli mitiores* (2) the cough, the frequent vomiting, and the heavy catarrhal symptoms which so generally attend the primary fever, render medical attendance necessary from the first moment of the attack. The treatment of these symptoms, however, and also of the eruptive stage, as long as the patient continues free from any serious

Wm. Johnston M - 11 Chd 1 yr old
M - x - xx - 3 1/2 Adult

inflammatory affection of the lungs, need not necessarily be active, it being sufficient to alleviate the cough, allay the vomiting, and check the catarrh by some of the large class of saline laxatives, lintseed tea, or mucilaginous mixtures, to which *antimonial wine* may be added, if necessary, as a diaphoretic, and to subdue high vascular action. In making a selection from these, the physician must be principally guided by the state of the bowels and the condition of the stomach of the patient. If the bowels be constipated, the milder purging salts, as the *sulphate of magnesia*, are to be preferred. On the contrary, if the patient be purged, and the vomiting distressing, a neutral mixture or effervescing draught will be found most beneficial. There are many persons in whom the cough and catarrh are the most urgent symptoms; and in such cases, if the stomach be quiet, the *liquor ammoniac acetatis*, in half ounce doses, combined with *camphor mixture*, from its more powerful action on the skin, is an excellent substitute. Another remedy, equally or perhaps still more useful, is *ipecacuanha*, of which from one to two grains may be given every four or six hours. Some practitioners prefer antimony to *ipecacuanha*, but antimony appears, at least in large doses, to act in some instances perniciously on the lungs.

The treatment which has been specified is, in most cases, all that is necessary throughout the whole course of the disease; and the greatly extended experience of Willan hardly enabled him to enlarge it. He was of opinion, however, that an emetic, given on the second or third evening, somewhat alleviated the violence of the catarrhal symptoms, and contributed to prevent the diarrhoea which usually succeeds measles. An emetic is especially useful if the disease be threatened with croup as a complication. During the eruption, he adds, "I have not observed any considerable effect from antimonials or other diaphoretics." Bathing the feet every evening seems a more beneficial application. Emulsions and mucilages afford but a feeble palliation of the cough and difficulty of breathing. With respect to opiates, they are not generally advisable: in the early stages especially, according to Willan, opium produces an increase of heat and restlessness, without conciliating sleep.

The catarrhal symptoms are frequently accompanied, even in the very earliest days of the disease, with much bronchial inflammation, and sometimes with pneumonia; or these affections may

occur at any later period, after the decline of the eruption, from the tenth to the twelfth day of the attack. Although experience has shown that bleeding may be practised with impunity in the very first onset of the disease, or at any subsequent stage, if the constitution of the patient is otherwise good, yet it is very rarely necessary to bleed before the subsidence of the eruption; for, if we wait that event, we "usually find the pulse become moderate, and the uneasy, laborious respiration terminate in twenty-four hours. This oppressed breathing is common to other eruptive fevers; and if it were universally to be considered as an indication for bleeding, the practice would often be more fatal than the disease" (WILLAN). If, however, pneumonia be threatened, blood should be freely but not extravagantly taken; for it should be remembered that although some children bear the loss of blood well, yet that others are long in recovering from it, even when the quantity drawn is small. In children, then, below ten years of age, when it is considered necessary to withdraw blood from them, it is more prudent to take blood frequently, and in small quantities, than in a large quantity at once. We should likewise be content with moderating the symptoms; for, as the inflammation depends on a morbid poison, it has a course to run, and does not admit of a cure. The bleeding should also be more moderate during the eruption than after it; for we have a right to look for a diminution of all the symptoms as the eruption naturally disappears. If pleurisy alone supervenes, leeches or cupping to the chest will be sufficient, without venesection. Blisters, ipecacuanha, and mercury are amongst the best *adjuvantia* to bleeding in severe cases. Mercury is best used in the form of friction with blue ointment over the chest, a little croton oil being added to promote its absorption (ANDREW ANDERSON). In cases where miliary tubercle may be suspected to grow, good results have been obtained from quinine, nutritious diet, and wine. In the Kiel epidemic already noticed, leeching and emetics, employed moderately, were unsuccessful; but the alternate application of towels dipped in hot and cold water had very good effects, but required to be continued for hours or days. The disease was very prone to relapse, and never pursued the typical course of a simple pneumonia.

Diet
During the whole course of measles it is necessary to enjoin an abstinence from all animal food, and to limit the patient to a

Milk, Beef tea
 low diet and to slops. The chamber should be of a moderate temperature (60° Fahr.), not subject to any sudden change from heat to cold, and the strictest cleanliness should be observed. With a view to protect the eyes, the room should be kept dark, so that the patient may be prevented reading, or using his eyes. In large establishments separation is necessary, to prevent spreading of the disease, if possible. Should the eruption disappear or be retarded, and untoward symptoms appear, such symptoms must be carefully studied, as prompt measures may be demanded, with the object of bringing the eruption "out," and subduing internal irritation or inflammation. A most efficient help is the hot or vapour bath. Warm drinks may also be given; and if there are no bronchial symptoms, or evidence of cerebral oppression, a dose of compound powder of ipecacuanha will be of service, proportioned to the age of the patient. *W*

When convulsions occur in children, hot foot baths sometimes give relief, as well as sinapisms to the limbs; after which, if they do not subside, blood must be taken by leeches from the temples; and it is in all cases necessary to determine the most probable source of the irritation, giving rise to the convulsions—*i. e.*, whether they depend upon the specific poison of the disease, upon dentition, or upon intestinal irritation or cerebral disorder. Diarrhoea should not be checked suddenly, but kept under control. *X*

Nov. 17. 69 Pinner

SCARLET FEVER.

LATIN, *Febris rubra*; FRENCH, *Scarlatine*; GERMAN, *Scharlachfieber*;
 ITALIAN, *Febbre Scarlatina*.

Definition.—*A febrile disease, the product of a specific poison, which is reproduced during the progress of the affection. On the second day of the illness, or sometimes later, a scarlet efflorescence generally appears on the fauces and pharynx, and on the face and neck, which spreads over the whole body, and commonly terminates in desquamation from the fifth to the seventh day. The fever is accompanied with an affection of the kidneys, often with severe disease of the throat, or of some internal organ, and is sometimes followed by dropsy. The disease runs a definite course, and as a rule occurs only once during life.*

Pathology and Symptoms of the Disease in its Varied Forms.—After a definite period of latency, the peculiar poison of scarlet

Eruption lasts 6-8 days
Sore throat 8-20

fever induces a disorder of the blood, which is, in the first instance, made manifest by a *febrile state* and a *disturbed condition of the great nervous centres*. The primary fever having lasted for one, two, or three days, does not entirely subside, but the secondary actions of the poison are set up as a peculiar eruption, preceded, followed, or accompanied by a sore throat. The eruption runs a course of from six to eight days, but the duration of the affection of the throat is more indefinite, and varies from eight to twenty, or more days. The fever continues during the eruption, and as long as the sore throat exists; but this being terminated, it subsides, and the disease is ended. In a few instances, however, tertiary results succeed, as dropsy or inflammation of the joints, diseases quite as formidable as any which had preceded them. As in ordinary fever, the poison of scarlet fever acts on the brain and its membranes, often causing the usual forms of inflammation of those parts, modified in their course and effects by the nature of the specific febrile disease.

That fever precedes the specific actions of the skin in this disease is so general a rule that it has few exceptions; and the pyrexia has been occasionally so severe as to destroy the patient before the more specific lesions of the disease have been set up. Dr. Andrew Anderson writes that he has seen death take place in six hours from the commencement of the disease—the child, in fact, dying poisoned (*On Fever*, p. 77). In suddenness of danger it thus approaches yellow fever and cholera. Again, the rule that the great specific action of the poison is expended on the skin, causing the specific eruption, has very few exceptions. Of this eruption there are several forms, such as *smooth*, *papulose*, *phlyctænoid* or *vesicular*. These are all evanescent after death.

In the smooth eruption the surface of the inflamed skin presents no inequality either to the sight or touch. The *scarlatina papulosa* has an eruption in which the papillæ of the skin are enlarged, and the appearance is that of roughness, or “goose-skinned.” The third form is when the eruption is accompanied by a number of vesicles filled with serum, which ultimately shrivel up and desquamate.

Whatever the ultimate form of the eruption may be, its first appearance is by innumerable small bright-red puncta, dots, or maculæ, separated by interstices of healthy skin. These puncta or maculæ are at first very minute points all over the affected

parts of the skin, which are usually more or less rough to the touch; but they quickly become confluent, so that in a few hours the redness becomes general over the parts attacked. The colour, in ordinary cases, is in the first instance a bright red, like that of a boiled lobster, but on the decline of the disease it becomes deeper, and more resembles that of beet-root, while in severe cases it is of a pink blush, rather than a scarlet efflorescence, or it may be livid, and intermixed with petechiæ. But whatever tint the eruption may assume, it has this peculiarity, that it disappears on pressure, and again returns from the periphery to the centre on that pressure being removed. The colour is also always brighter and more vivid in the flexure of the joints, and about the hips and loins, than over the rest of the body. A sign of scarlatina, in connection with the eruption, has been described by Bouchut, as pathognomonic. It consists in an enduring white stripe, produced by pressure with any hard substance on the skin occupied by the eruption. This phenomenon is ascribed to an increase of the contractile power of the capillaries, and which is proportionate to the intensity of the disease, the regularity of the eruption, and the amount of vital power (*Syden. Society Year-Book*, 1861, p. 130). The termination of this inflammation is generally by desquamation of the cuticle, and the desquamation begins with the decline of the eruption; and is usually completed by the end of the second week, unless it is longer delayed by successive crops of eruption, and consequent succession of exfoliations. There is no fever in which this phenomenon is more conspicuous. A few days after the commencement of the desquamation, albumen may be detected in the urine in small quantity, which continues to be given off for several days, along with a considerable amount of epithelium from the uriniferous tubules (DR. J. W. BEGBIE). There are also cases in which the albuminuria is associated with *anasarca*; and three stages have been recognized in which this complication occurs:—(1.) A febrile stage, characterized by fever of an intermittent character, and by rapid serous extravasation and infiltration; (2.) A chronic stage, in which the affection follows a slowly progressive course; (3.) A period of resolution (HAMBURGER). Such cases sometimes terminate by uræmic symptoms and convulsions. An unusual case of this nature has been recently recorded by Biermer. It happened with a boy five years and a half old, and ended fatally on

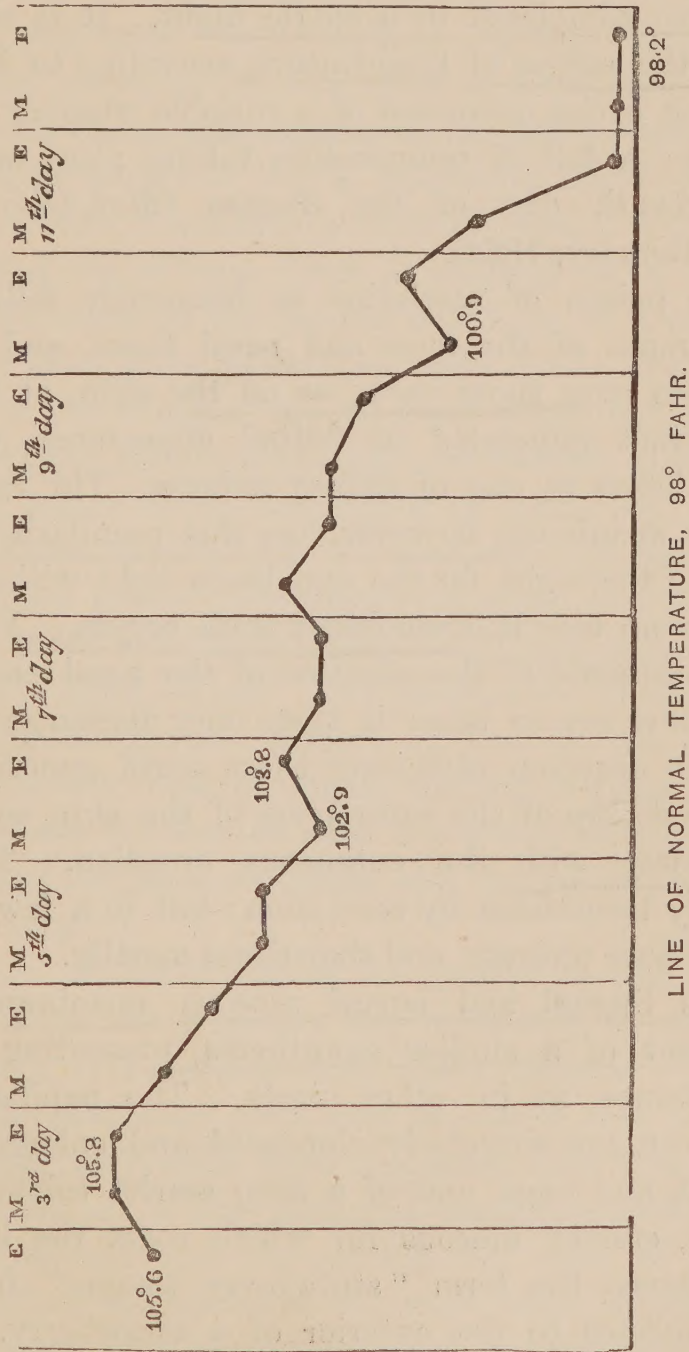
Temp may rise to 105 - 112

the thirty-fifth day. No urine was passed for 108 hours between the twenty-first and the twenty-sixth days of the disease, and extremely little for five days more. Yet during these ten days there were no uræmic symptoms, nor any notable dropsy. The uræmic symptoms first set in after the urine began to be secreted freely, and it was but slightly albuminous (*Syden. Society Year-Book*, 1861, p. 218). Occasionally the *squamæ* of the cuticle are so large as to preserve entire the whole epidermis of the palms of the hands and of the soles of the feet. Frequently, however, the material of desquamation is furfuraceous or scaly. Frank has seen the cuticular *squamæ* come away with the hair, nails, and even with *veruæ* attached. In a few instances the termination is by ulceration and sloughing of large portions of the integument.

Whatever may be the colour or description of the eruption, it does not attack all parts of the body simultaneously, but appears partially, or in a succession of crops, the order of which may be stated as follows:—On the first day it spreads universally over the face, neck, and upper extremities; on the following day over the trunk, but is less general on the back than on the abdomen; and, lastly, on the third day, it has extended itself over the lower extremities. The duration of each crop is about three days, when it disappears, and in the order of attack, falling from the head and upper extremities on the fourth day; from the trunk on the fifth day; and from the lower extremities from the sixth to the eighth day. The order of attack, however, which has been mentioned is not constant, for in some few instances the eruption appears first on the trunk and lower extremities, and only on the second day very faintly on the face and upper extremities. The disease attains its height, and the fever maintains its course, usually from the fifth to the ninth day, when, in favourable cases, continuous defervescence sets in, and all the symptoms begin to decline. The fever does not subside on the appearance of the rash, as is the case with small-pox, but continues, with various degrees of violence and ranges of temperature, throughout its progress. The pulse is often 120 to 130 in a minute, and sometimes beats with considerable force. The skin frequently indicates by the thermometer a temperature of 105°, 106°, or even 112° Fahr.; and it is dry, with a sensation of burning heat till about the third day, when the maximum of temperature is attained. From the third to the ninth day the range is main-

tained between 103.8° and 102.9°, and begins to subside about the tenth or twelfth day, after which the defervescence is continuous. The difference in these respects between scarlatina and measles may be appreciated at once by a glance at the account and the diagram given of measles, and comparing it with the following, which shows

THE TYPICAL RANGE OF TEMPERATURE IN A CASE OF SCARLATINA. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE SECOND DAY (Wunderlich).



There is no remarkable increase of fever heat preceding complete defervescence; and after the *exanthema* has reached its maximum, the decrease of temperature proceeds by no means

Cycles of 5 days temp

rapidly. The commencement of the decrease may be marked by a few decided and rapid falls; but its farther fall is decidedly lingering, and is even sometimes interrupted by small increases of temperature, so that the whole process of defervescence occupies, as a rule, from five to eight days. It is only in very mild or anomalous cases that the temperature rarely exceeds 101.8° Fahr.; and these cases sometimes show a rapid defervescence, completed in a single night. It is essentially a short fever, the ranges of temperature, according to Dr. Sidney Ringer, forming cycles composed of a variable number of days, generally of five; a fall of temperature taking place on the fifth, tenth, or fifteenth day of the disease (*Med.-Chir. Society Trans.*, 28th January, 1862).

The poison of scarlatina as frequently falls on the mucous membranes of the eyes and nasal fossæ, and excites a similar eruption over those parts, as on the skin, at first consisting of a distinct punctated or dotted appearance, which changes in a few hours to one of diffuse redness. The inflammation of the ocular membrane, however, has this peculiarity, that it does not distress the sight, for the eye bears light without inconvenience, and in no case is it combined with coryza. Neither is sneezing a consequence of the affection of the nasal membrane; and only in a few severe cases is there any discharge from the nostril. As the eruption attacking these parts generally appears with the first crop of the *exanthema* of the skin, so does it generally die away with the cutaneous eruption. This inflammation usually terminates by resolution; but in a few instances the alæ of the nose ulcerate, and sometimes mortify.

The lingual and buccal mucous membranes are also often the seat of a similar exanthema, presenting nearly the same appearance as in other parts. The papillæ of the tongue, however, are singularly elongated and enlarged, and stand up salient and erect, and of a deep scarlet colour, above the thick, white, creamy mucous fur which coats the lingual membrane; and hence the term "strawberry tongue," from the supposed resemblance to the exterior of a strawberry. The tip of the tongue is of a vivid red, through development of the papillæ. By and by the fur falls off, and the whole dorsum of the tongue is then left clean, red, and raw-looking. This affection lasts longer than that of the eyes and nose, and usually terminates

Scat to favour Tonsils less swollen, more hard, more
deeply ulced, longer in healing

AFFECTIONS OF THE MUCOUS MEMBRANE IN SCARLET FEVER. 325

by resolution, although, in a few instances, the buccal membrane ulcerates and mortifies.

The sore throat, or inflammation of the faucial membrane, though not so constant an affection as that of the skin, yet, when it does exist, it is often of much longer duration, and is a much more grave disease. It may either precede all the other symptoms, or it may occur at any period of the fever. This inflammation, at first punctated, then diffused, usually runs into ulceration, and the character of the ulcer is so completely in unison with the state of the constitution as to enable us, according as it is slight or severe, to divide scarlatina into two great varieties—namely, into “*scarlatina mitior*” and “*scarlatina gravior*.” The first, or sthenic form, is marked by a greatly enlarged or swollen state of the tonsils, which are of a vivid or bright-red colour; and, when ulceration takes place, the ulcers are seldom deep, or the sloughs slow to come away, but usually they separate about the fifth or sixth day; so that in mild cases the sore throat is healed about the eighth or tenth day, and in more severe ones about the fifteenth or twentieth. In malignant cases, or in *scarlatina gravior*, the tonsils are much less tumefied and enlarged, but much more loaded with blood, and of a deeper, and sometimes of a livid colour. The ulcers, also, are deep and formidable, and the sloughs are thrown off later in the disease. They are likewise slow to heal, or not till the end of three weeks, and in severe cases not till four or even six weeks have elapsed, during which period the fever continues, and the patient remains in considerable danger.

The inflammation of the throat is not limited to the tonsils, but extends over the fauces generally and uniformly, or symmetrically on either side, as is common with blood diseases. It may extend to all the neighbouring parts, and an abscess may form in the pharynx, or pus may issue from the ears. The tympanum has been eroded, and in a few instances the inflammation has extended to the larynx, and the patient has died of croup. Besides these disorders, the glands of the neck often enlarge and occasionally suppurate, and, singular to say, sometimes not till after the sore throat has healed, and sometimes when there has been no previous affection of the throat, as if these parts were the seat of a specific action of the poison. Glandular swellings on both sides of the neck are not unfrequent, and have been

Otitis. Pharyngitis - gland swellings in neck
Dropsy here appears from 15-25th day

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described as "the scarlatinal bubo" (TROUSSEAU). They may be the best and most accurate index of danger in the later period of scarlatina, in so far as the danger depends on the sore throat, and on the putrid infection of the blood (septicæmia) which accompanies it (W. T. Gairdner, *Clinical Medicine*, p. 193).

swelling of
face
The inflammation of the cutis, as also of the buccal mucous membrane, is usually accompanied by some inflammation of the submucous or subcutaneous areolar tissue. This affection takes place as soon as the rash appears, and causes the hands to swell, so that the patient is unable to bend his fingers, and his face becomes tumefied and painful. In mild cases, however, the serum effused is absorbed, and the disease terminates without any unpleasant consequences; in severe cases it has a tendency to terminate in ulceration or in mortification. In children the toes of one foot have been known to slough off; and in some the integuments of the leg have mortified from the knee to the foot; while in others, mortification, commencing in the upper lip, has been known to spread till one-half the cheek was eaten away. Some have been known to die of mortification of the rectum, and others of a similar affection of the pudenda.

Such are the primary and secondary affections of scarlatina; but this poison has also some tertiary actions, giving rise to dropsy, as well as affecting the synovial membranes of the joints.

The dropsy which sometimes occurs after scarlet fever usually commences between the fifteenth and twenty-third days of the disease, and, almost uniformly, not till after all the other symptoms have subsided. The patient is liable to it during desquamation, as already mentioned, and for a considerable time afterwards. It begins with anasarca of the face, and afterwards of the hands and feet. In some instances the anasarca is universal, the whole areolar tissue of the body filling so rapidly as sometimes to destroy the patient in a few hours, the cavities of the chest and abdomen frequently filling at the same time. According to the observation of Dr. Wood and many others, it has occurred more frequently after mild than severe cases. Its forms are, therefore, *anasarca*, *ascites*, *hydrothorax*, *hydro-pericardium*, and even *hydrocephalus*; but, in whatever form, heaviness approaching to stupor is a common attendant. During the progress of the fluid effusion, oedema of the glottis must be watched for and relieved. The dropsy is generally accompanied with scanty and albuminous

Case Phlegmonous Lymph - left side face
reaching up to scalp & down over shoulder
1 wk of this for child 10 months - death Feb 73

urine; and although the presence of albumen, without diminished secretion, is almost a regular phenomenon in the course of the disease, independent of dropsy, as shown by Dr. James W. Begbie, yet, if the urine becomes highly albuminous and diminished in quantity, the dropsical complications may be apprehended (*Edin. Med. Journal*, Jan., 1849, and Oct., 1852).

More or less congestion of the kidneys occurs in every case of scarlet fever, although, like the sore throat, it may often be so slight as not to give rise to any prominent symptom (BEGBIE, ANDERSON). The scarlatinal dropsy is very generally considered as most intimately connected with the kidney disease; and when the kidney disease is well marked, the characters of the urine exactly resemble those in acute Bright's disease (PARKES). On the other hand, there is also evidence decidedly in favour of the opinion that albuminuria may be wanting in scarlatinal dropsy (See Parkes *On the Urine*, p. 264).

The condition of the urine in scarlet fever ought to be ascertained daily in every case, especially during the period of convalescence. "It is of more importance," writes Dr. Andrew Anderson, "that you should examine the urine than that you should feel the pulse of a convalescent from scarlet fever." The urine has the ordinary febrile characters. During the first six days the amount is small; the urea and uric acid are increased in amount, and sediments of urates occur. The chlorine is sometimes greatly lessened, and augments during convalescence. On the sixth to the eighth day, if the case goes on well, the urine becomes abundant, pale, and the re-action neutral or feebly acid. There is bile-pigment present during the first six days; and in a large proportion of cases, though not in all, the urine becomes albuminous. Dr. Warburton Begbie believes it to be present at some period in almost every case. It is usually associated with a large amount of renal, pelvic, and bladder epithelium, but not with renal cylinders (BEGBIE), unless there be dropsy. The albuminuria occurring during desquamation is usually transient; but it may continue till an attack of dropsy occurs—disappearing and re-appearing, when dropsy comes on a fortnight or three weeks later. In malignant scarlatina, as in malignant variola, there may be considerable hæmaturia or passage of dissolved hæmatin (Parkes *On the Urine*, p. 263).

Intercurrent inflammations of the synovial membranes have

been described by Withering, Sennertus, Heberden, and others. This disease may attack the wrist, ankle, or knee-joints, and usually terminates by effusion of serum; and in some cases the cavities of the joints contain pus. This inflammation seldom occurs till after the eruption has subsided, and is generally a tertiary phenomenon in the course of the specific disease.

Such are the morbid phenomena which have been observed in the ordinary course of scarlatina, and with sufficient constancy to mark the disease as due to a specific poison; but these appearances are only to be found when the disease is of moderate intensity and the patient survives some days. In severe and rapid cases the patient may die, not from any organic lesion, but from the intensity of the shock, in the first instance, on the nervous system; for Bretonneau, Tweedie, and Sims, all speak of having examined the bodies of persons who have died early in the disease, in which there was scarcely any appreciable lesion—coma, or other violent cerebral affection, carrying off the patient.

Although several varieties of scarlatina are described by authors, it is not to be supposed that they are equally distinctly defined in nature. Yet it not unfrequently happens that the characters of each variety are tolerably well marked. The following may be distinguished, namely:—(1.) "*Simple Scarlet Fever*;" (2.) "*Anginose Scarlet Fever*;" (3.) "*Malignant Scarlet Fever*;" and (4.) "*Latent Scarlet Fever*."

Scarlet fever, of whatever description, essentially consists of fever, already described, and certain local inflammations; but among its more striking phenomena is the sudden and remarkable depression of the mental and physical powers of the body which the poison produces—a depression so great as sometimes to cause the death of the patient in a few hours, without any re-action, or any very sensible local lesion of the throat or other part being discoverable after death. On the contrary, there are a few instances in which the re-action is so great as to destroy the patient in an equally short time, and with a similar absence of all pathological phenomena: the affection of the skin being suppressed, and the sore throat wanting, the patient dies as if from the influence of an overwhelming poison.

The symptoms of scarlet fever under ordinary circumstances may be divided into three stages. The first stage occupies the period from the commencement of the disease till the appearance

of the eruption, and is technically termed the "*primary fever*;" the second stage, that from the appearance of the eruption till its entire subsidence; while the third stage is reckoned from the disappearance of the eruption till the termination of the disease. The duration of the first stage is one, two, or three days; that of the second from six to eight days; while the third stage may either not exist, or vary from a few hours to two or three weeks, making the whole duration of the fever to vary from eight to thirty or more days. These stages are not, as in typhus, usually marked by changes of the tongue; for, except in the more severe forms of the disease, it continues coated with a white creamy mucus throughout the whole course of the disease. In "*scarlatina anginosa or maligna*," however, it becomes brown or black in the second, or at the commencement of the third stage.

The primary fever may be sudden in its attack, or the patient may complain for some days of slight indisposition. The early symptoms, whatever be the variety, are headache, pains in the back and loins, loss of appetite, sickness, and white tongue. The disease is, indeed, usually ushered in by vomiting—sometimes by very obstinate and troublesome vomiting. In slight cases it is sometimes the only noticeable symptom (ANDERSON). Still there are symptoms which distinguish it from other continued fevers; for the pulse, instead of being full and strong, is small and weak and rapid; the heat of the skin is more ardent; and, with such ranges of temperature as have been already noticed, these phenomena continue through the whole course of the disease. The fever, however, varies greatly in intensity, as already indicated, from a mere febricula to the severest forms of a typhoid type in protracted cases.

1. *Simple Scarlet Fever.*

This form is known by the name of *S. mitis* and *S. sine angina*. It is the simplest form of scarlet fever, and is limited to cases with the fever and eruption, without any affection of the throat, or to cases with "a scarlet rash, with redness of the throat, but without ulceration."

The symptoms of this variety are extremely mild, so that the patient is frequently not confined to bed. The primary fever, except that the pulse is rapid, is little more than a mere febricula, and is not aggravated on the appearance of the eruption. The eruption appears at the end of twenty-four or forty-eight hours,

1st Stage 1-3 days
2d 6-8 days
3rd variable a few hrs to 2 or 3 weeks

and the crops follow each other according to the usual order of succession, appearing first on the face and neck and upper extremities; on the following day on the trunk; and on the third day on the lower extremities, when the disease has reached its acmé. On the fourth day the rash begins to decline, and fades from the face, neck, and upper extremities; on the fifth day it disappears from the trunk; and on the sixth or seventh day it is evanescent over the whole body. The colour of the rash is always more florid during the night than in the day; and on its declining, desquamation takes place. With the disappearance of the rash the fever of this variety ceases, and the disease terminates; but it often leaves the patient in a state of considerable debility for several days, and may be followed by albuminuria.

2. Anginose Scarlet Fever.

*Felix sine
eruptione*

In this form of the disease the specific action of the poison is mainly limited to one region—that of the throat—the eruption on the skin being altogether wanting, or appearing at a later period than usual, generally by one day; and, as a general rule, is less copious and less diffuse than in the other forms. It is “a more severe form of the disease, with redness and ulceration of the throat, and a tendency to the formation of abscesses in the neck.”

There is seldom a season in which scarlatina has been in any degree epidemic, that cases have not occurred in which patients (not having previously had scarlet fever) are seized with severe fever and sore throat, unaccompanied by any eruption, and who, on subsequent exposure to the contagion of scarlatina, have been found insusceptible of the action of the poison. Hence it is inferred that the disease they have passed through must have been a variety of scarlet fever, or *scarlatina sine eruptione*, making itself manifest by a peculiar sore throat, associated with the febrile phenomena.

This disease, therefore, essentially consists in fever and sore throat. It has been stated that the state of the throat is constantly in unison with the state of the constitution, and consequently this form of disease, according to its severity, assumes all the symptoms which accompany scarlatina simplex, or the more severe forms, with the exception of the absence of the eruption. It seems unnecessary, therefore, to give a separate detailed account of this variety.

In its milder form the essential character is, that the secondary or specific actions of this poison fall on two tissues—on the skin, and on the mucous membrane of the eyes, nose, mouth, and fauces. The fever which precedes the eruption in such cases lasts from twenty-four to seventy-two hours. The symptoms, however, are more violent than in the preceding species; for nausea or vomiting, great restlessness, headache, and some delirium frequently occur as early as the second day. The heat of the skin also is more considerable, and often raises the thermometer as high as 105° , while the pulse is quick, feeble, and fluttering, and shows the extreme debility the poison has occasioned. The primary fever having lasted its period, the specific actions of the poison are set up, and the eruption runs the course which has been described in scarlatina simplex, but its colour is more intense, its duration more variable, and its attack more partial.

The *angina*, so marked a symptom in this affection, may precede the primary fever, may commence with the eruption, or may occur at some later day in the disease. It has many grades, and in this form of scarlatina they are all of the sthenic or inflammatory type. Thus, in slight cases, the throat has merely the sensation of roughness, with some pain in deglutition; at a higher degree the tonsils are enlarged and ulcerated; while in cases of still greater severity they are swollen to a degree almost to occlude the fauces. In this latter case the act of deglutition is not merely painful, but in many instances impossible, and is impeded by a thick viscid mucus, which frequently requires the effort of vomiting to remove. The irritation of the fauces is sometimes propagated to the larynx, and the patient's voice is hoarse or inaudible, and perhaps he may ultimately die from this new affection. The parotid and submaxillary glands often enlarge, sometimes previously to the sore throat, more commonly about the fifth day, and again after the sore throat has healed.

The degree of fever is usually proportioned to the severity of the *angina*, and is accompanied by headache and sometimes by delirium. It does not abate on the appearance of the eruption, but continues till the throat is healed. If the sloughs come away early, or on the fourth or fifth day, the throat heals, and the fever perhaps subsides within a day or two after the eruption. It sometimes happens, however, that the sloughs do not separate till

Fever continues till throat healed.

the fourteenth or fifteenth day; and in this case the fever runs on with equal violence after the disappearance of the eruption, and the whole disease is sometimes prolonged for three weeks or a month. In this case the tongue may become brown or dry, but it seldom continues so for more than a few hours. Observations as to temperature ought to be regularly and continuously made.

In the more severe forms of the *scarlatina anginosa* (and which have been described by some authors as the "*scarlatina gravior*"), the specific actions of the poison are the same as in *scarlatina mitior*, but the symptoms, both local and general, are more severe, and the tertiary affections more frequent, and, consequently, the disease is more grave and the danger more formidable.

The more remarkable symptom which distinguishes this form of the disease is the state of the tonsils. In the milder form previously noticed, it has been stated that the tonsils are either slightly affected or greatly enlarged, of a bright red, and the ulcers comparatively superficial; but in this severer form the tonsil, though less swollen, is more gorged with blood, more livid in colour, while the ulcers are foul, deep, and burrowing; the secretions of the mouth are more copious, and generally impregnated with the offensive sordes of the sloughs: while deglutition, if less difficult, is perhaps infinitely more painful, and the mouth often so tender that the slightest touch excoriates it. The ulcers likewise are slow to granulate, and only heal after a tedious treatment; and in the worst cases they spread in every direction, the parts tending to vesicate and even to mortify previous to the death of the patient.

The eruption offers some peculiarities, being often later by some hours in coming out, its colour darker and more livid, its duration more uncertain, and its distribution more irregular and capricious than in the milder form. The primary fever, likewise, is usually longer, the delirium earlier, and the depression more complete than in the milder forms; and towards the close of the disease the tongue becomes brown, and the symptoms closely resemble those of the last stage of typhus fever.

Such are the more marked characters of the severer form of scarlatina; but it often happens that the progress of this disease (unless the range of temperature is regularly and continuously recorded) is silent, slow, insidious, scarcely marked by any pro-

28/960/38
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minent symptom, till the degree in which the constitution is subdued by this formidable poison is shown by the inflamed nasal membrane discharging its fetid ichor, causing mortification of the alæ of the nose, or mortification of the lip or cheek; or it seizes on some remote part, as the toe, the leg, or the whole of a lower extremity, and which, for the most part, terminates the life of the patient. It may pass into the next form of the malady, namely, —

3. *Malignant Scarlet Fever.*

This form is that which is known as the “malignant sore throat,” or “putrid sore throat” of some authors; and is the name now generally applied to certain cases of extreme severity, into which some of the forms already described may pass, as if by insensible gradations. In this variety “the throat tends to slough; the scarlet rash is scarcely, if at all, visible, petechiæ are often seen on the surface, and the fever is of a low form.” In others, the violence of the attack is so sudden that the patient is at once struck down by the force or virulence of the poison, the type of the attack being at once septic, adynamic, typhoid, and malignant. The extreme severity of the constitutional symptoms is marked by the smallness, feebleness, and irregularity of the pulse; the oppressed, short, and quick respiration; the appearance of early raving, stupor, and sometimes coma, alternating with fretfulness and violence, dulness, and suffusion of the eyes, flushing of the cheeks, and dark-brown furred tongue. The rash appears late, and is of uncertain duration, and soon assumes a dark or livid colour, or disappears in a few hours, re-appearing again after several days, if life is so far prolonged. Aphthous elevations in the throat, surrounded by a livid base, also become dark, and, bursting, they expose a surface of an excoriated, dark, gangrenous appearance. The passages of the fauces are always clogged up with much viscid mucus or phlegm, which produces a rattling noise in breathing, and increases the pain and difficulty of swallowing. The discharges, often sanious, are remarkably acrid, which issue from the nostrils and posterior nasal passages, causing soreness, excoriations, and even blisters on the surfaces and orifices over which, or through which, they flow. To this source the diarrhoea may be ascribed, which is sometimes severe at this period, and generally adds greatly to the sufferings of the patient.

The severity of the symptoms may produce death on the second, third, or fourth day of the disease, as from gangrene occurring in the course of the œsophagus or alimentary canal. In other instances in which the early symptoms were not remarkably severe, the aphthous state of the throat has all at once assumed a sloughing aspect, and has carried off the patient at the close of the first week. When the disease is continued beyond this time, death is foretold by the rapid, small, and weak pulse; by the rapid, languid, and oppressed respiration; frequent fluid acrid discharges issue from the bowels, and blood may be discharged from the nostrils, mouth, throat, bowels, or even from the kidneys; petechial or purpuric spots appear on the skin; and the patient is at last destroyed with local manifestations of the morbid state in several different parts and organs.

4. *Latent Scarlet Fever.*

VB The marked prevalence of anasarca in children has led to the discovery that such children have had previous attacks of scarlet fever, in such a mild form that it has escaped detection. In such cases the constitutional affection of scarlatina has been produced, but without any rash or sore throat being observed. On the kidneys alone the poison makes itself felt, and the dropsy which ensues is more severe, complicated, and fatal than that which follows the regular forms of the disease.

Sequelæ of Scarlatina.—Under this head it is proposed to notice what may be called the “tertiary actions of the poison.” The effects produced in this way are often called by the people the “dregs” of the fever. The principal source of some of these sequelæ is found to be the primary obstruction to which the functions of the kidneys are so liable.

Antis Amongst the most important of these sequelæ are the effects produced by an extension of the original affection of the throat towards the internal ear, by the Eustachian tube. When this takes place it not unfrequently happens that the small bones of the ear are completely destroyed, the tympanic cavity becomes inflamed, ulceration of the membrane takes place, and perforation follows.

This morbid state is most difficult to remedy: a chronic discharge from the ear is established, which is of the most offensive kind, and which may continue till the whole of the internal ear

Dropsy here occurs from 22-23rd day

is involved in the destructive and inflammatory processes; till the delicate and soft tissues in the cochlea and semicircular canals are destroyed, and the petrous portion of the bone itself dies; till the mastoid process, with its capacious osseous areolæ, becomes the seat of an obstinate carious process; or even till the brain itself, or the membranes, are involved in the unhealthy inflammatory process. Such a combination of effects occasions great and protracted sufferings, and sometimes in the end a fatal result (BRUCE, ANDERSON). A similar inflammation may destroy the tissues in the back part of the pharynx, extending towards the base of the cranium and upper cervical vertebræ.

A frequent form in which the tertiary actions of the poison of scarlatina are manifested consists in inflammation of the joints, and dropsy; and it is singular that these diseases are more often set up after mild than after the more severe forms of this fever. In such cases, about the time of the disappearance of the rash, the joints of the wrists or fingers, of the knees or other articulations, become swollen and inflamed, and present all the phenomena of an attack of acute rheumatism. This affection keeps up the fever, and prolongs the whole duration of the disease for many days beyond the usual period.

Again, in a given number of cases, not exceeding three *per cent.* in general, but in different seasons, or under different treatment, sometimes amounting to twenty *per cent.*, the tertiary action of the poison produces dropsy. This affection usually occurs about the twenty-second or twenty-third day, or about the time when the patient is convalescent, and more often after a mild than after a severe disease. Dropsy more commonly begins with pallor of the countenance, and with oedema of the face; then the hands and feet swell, and, in a few cases, the areolar tissue of the trunk and lower extremities becomes enormously distended. When the areolar tissue is thus slightly or more generally distended with fluid, effusion may take place into the cavities of the head, chest, or abdomen. When the brain is threatened, the effusion is commonly preceded by the usual hydrocephalic headache, by convulsions, and sometimes by blindness. Effusion into the cavity of the chest or of the abdomen causes the usual symptoms of hydrothorax and of ascites, which have been described. In the former instance, however, the

Scarlet Rh

M

vide Vol II

Time of life most liable 3-4th years
Season Oct - Dec

watery fluid is sometimes poured out so rapidly as to destroy the patient in a few minutes or in a few hours.

The first appearance of the œdema or effusion is usually preceded or accompanied by an accelerated pulse, by the urine being scanty, commonly turbid, and passed with pain: the quantity, however, is shortly increased; and if examined when passed copiously, it is found to be of low specific gravity, or from 1.011 to 1.017, and to contain albumen, sometimes blood, renal epithelium, and cylinders.

Diagnosis.—The only diseases with which scarlatina can be confounded are the acute forms of roseola and measles. Roseola, though usually accompanied by fever and sore throat, is distinguished from scarlatina by the eruption being confined generally to the chest. The diagnosis between measles and scarlatina will be better understood after the next disease we have to notice has been described—namely, the hybrid form sometimes assumed by a concurrence of the two diseases; and by a careful study and observation of the ranges of temperature in each.

Cause and Propagation of the Disease.—The earliest source of the poison is distinctly traceable to Arabia; and the disease has now spread over the whole world. It prevails at all seasons of the year, is always in existence somewhere, and often epidemic. Scarlet fever has been found to spread more extensively, and with greater fatality, among the poorer than among the wealthier classes of society. Both sexes are attacked in nearly equal proportions. All ages are probably liable to the disease; but it is most common to childhood—the feebleness of this early period of life facilitating, perhaps, the reception of the poison; and as children grow older, the less liable are they to be attacked.

In a clinical essay on the *History of Scarlet Fever*, most carefully worked out by Dr. B. W. Richardson, it is shown that scarlet fever attacks most frequently in the third and fourth years of life. The chances of attack decline rapidly after the fifth year. The seasons also seem to influence its prevalence and intensity. The months of October, November, and December, furnish in England the maximum amount of the disease—the months of April, May, and June, the minimum. This disease being established, the patient generates a poison which may be communicated directly, or which may contaminate the atmosphere. The disease is eminently communicable, so that no susceptible person

Second attacks rare
May coexist with other diseases

can remain in the same room, and hardly in the same house, without contracting it. The *infecting distance* is consequently much greater than in typhus. Indeed, it is necessary to break up every academic establishment in which scarlatina prevails; for it is hardly possible to isolate children in the same house or school, however large, so as to prevent the disease from spreading. That scarlatina is capable of being directly communicated is shown by the fact that children have been inoculated with the serum found in the vesicles which sometimes accompany the rash, and have taken the disease; but the inoculated disease not having proved milder than the natural, the practice has been properly abandoned. Another proof of the directly communicable nature of scarlatina is, that it has often been propagated by *fomites*, as by the clothes and boxes of boys returning from school. Susceptible persons also sleeping in a room lately occupied by patients labouring under scarlatina, and before the furniture has been washed and the bedding and walls well ventilated, have often taken the disease. The virus is destructible by heat at the boiling point, or it may be disinfected artificially, as by the fumes of nitrous acid.

Dr. Willan says, that out of 2,000 cases that he attended, he witnessed no instance of a *second attack*. Still, there are some exceptions to the statement that an attack of scarlatina gives an immunity from a second attack. Dr. Binns has seen instances of scarlet fever occurring twice in the same person, while Sir Gilbert Blane met with an instance of its occurring thrice in a young lady, without the least suspicion of ambiguity or possibility of mistake in diagnosis. Dr. B. W. Richardson shows that it may recur once or even twice in the same person. But these events are rare; and death from a second attack is unknown as a fact.

Scarlet fever has often co-existed with the vaccine disease, and with erysipelas, and this poison is consequently capable of co-existing in the system, not only with those that have been mentioned, but probably with all other morbid poisons.

The poison of scarlatina is absorbed by the mucous membranes; and absorption is also evident from the fact of inoculation having been effected through the skin. Children have been born labouring under this disease.

The period of latency varies from a few hours to ten days. In one case inoculated by Rostan the disease appeared on the

seventh day; and the specific poison is probably capable of communication from the patient to others as soon as the primary fever has formed, and perhaps continues to be so till the sore throat has perfectly healed, supposing that affection to continue after the eruption has died away.

Prognosis.—The mortality from scarlet fever varies greatly according to the season, and also, perhaps, according to the fatality of the epidemic. In some years the proportion of deaths is not greater than three *per cent.*; but Sir Gilbert Blane says his practice gave one in four. He was consulted probably only in the worst cases, for in the same year it appears, from the reports of other practitioners, the deaths varied from one in six to about one in thirty.

There is perhaps no disease in which the progress is so capricious: for it is found to vary with the several forms, types, complications, epidemic constitution, and with the treatment in a most remarkable degree. The mortality is greatest in the period of infancy and childhood—from one to five years. In relation to mortality, it seems second in this country as to severity, typhus fever standing first (RICHARDSON). It is twice as fatal in towns as in the country. "There is one condition in which the disease is almost invariably fatal; that is the puerperal state." No precaution ought, then, to be neglected, no precaution ought to be thought excessive, which tends to prevent a woman from receiving the poison of scarlatina while pregnant or recently delivered" (DR. ANDREW ANDERSON). Fever during the pregnancy most certainly ends in abortion and death. If the woman be recently delivered, the disease will be of the most malignant type, and almost always fatal.

Treatment.—Scarlet fever being evidently accompanied by many highly inflammatory symptoms, the practice of bleeding was adopted on the first breaking out of the disease, in all countries, and, according to Willan, *with the most disastrous results*. This mode of treatment was adopted by Morton; and he speaks of witnessing 300 deaths from scarlatina in a week. It prevailed down to the time of Huxham, who abandoned it, and introduced a treatment by bark. In this manner an entirely opposite system of treatment has been gradually introduced, and the records of medicine enable us to state the results of these opposite modes:—Of cases treated at the Foundling Hospital by

bleeding in 1786, and of cases treated at the London Fever Hospital in 1829, in the same manner, it seems proved that one in six died after bleeding, while only one in twenty-two died after a milder, if not a directly opposite, mode of treatment; and the conclusion which inevitably follows is, that the chances of recovery are diminished by the practice of bleeding, nearly in the ratio of four to one as compared with the chances of recovery supposing the patient not to have been bled.

It remains now to give some general directions for the treatment, and to point out the circumstances in which bleeding, purgatives, wine, and tonics may be most advantageously employed.

It should be laid down as a maxim, that in scarlatina, medical advice ought always to be had recourse to; for the worst cases we meet with (as those in which mortification of the nose, cheek, or limbs sometimes takes place) are those in which the disease has, from its apparently mild character, been left to itself. X NB

In the mildest form of the disease it is sufficient to confine the patient to the house; to enjoin strictly a milk diet; to regulate the bowels; and, above all things, to avoid the *nimia diligentia medicorum*. If anything more be done, a small quantity of wine and water, proportioned to the age of the patient, may be given.

A gentle emetic at the outset is believed to have a happy effect in modifying the future course of the disease. Ipecacuanha, with or without tartar emetic, is the best form for administration; and half an ounce of castor oil ought to be given after the action of the emetic had ceased.

Looking to the morbid condition of the blood, and to the tendency which exists to the deposition of fibrine in the right cavities of the heart, small doses of *carbonate of ammonia* (three to seven grains) administered every hour, or every three hours, as soon as the symptoms are decided, have been recommended (PEART, WITT, RICHARDSON). Or, the *liquor ammoniæ acetatis* may be used with an excess of ammonia, to the amount of from three to five drops of *liquor ammoniæ* added to two fluid drachms of the former in a liberal quantity of distilled water (RICHARDSON). It is important to administer these medicines in small and frequently repeated doses; and, if possible, to let the remedy be taken as a drink.

The treatment of the milder forms of the fever, when the tonsils are considerably enlarged, is first to tranquillize the

stomach and allay its perverted action when vomiting exists, either by small doses of the sulphate of magnesia, or by the effervescing draught,—medicines which, according to the state of the bowels, may be given every four or every six hours. The gum resin of guaiacum is of great service in subduing the cynanche tonsillaris, and may be prescribed in the following formula:—

R. Magnes. Sulph., ℥vi.; solve in Aqua, ℥viii.; adde Pulv. Guaici, ℥iiss.; Pulv. g. Tragacanth. co., ʒij.; *misce bene*. One-sixth part of this mixture may be given every four hours, till the bowels are freely moved. r XV

As soon as this object is effected, and it is ascertained that the tonsils are still greatly enlarged and swollen, the practice (supposing the patient to be *an adult*) is to relieve them by the application of six to twelve leeches to the throat; and the bleeding may be further encouraged by the application of a poultice. The trifling loss of blood thus sustained does not impair the general strength of the patient, if it is done sufficiently early, while it greatly reduces the swelling of the tonsils, and may prevent them becoming permanently enlarged. Another advantage is gained by the application of leeches to the throat—namely, that they relieve the affection of the head; for we constantly observe that, in diseases depending on morbid poisons, the head symptoms are relieved by relieving the part specifically acted upon.

The tonsils having been thus relieved, the fever ought to be permitted to run its course uninfluenced by medicine, the patient being only refreshed by the occasional administration of the saline draught, so grateful to his parched mouth and feverish state. If stimulation be adopted in these cases, we are apt to bring back the tumefaction of the tonsils; while, on the contrary, if we take more blood, we hazard producing the more serious accidents incident to scarlatina. The medicines, therefore, that have been mentioned should be persevered in till the disappearance of the eruption, and till the healthy granulations of the throat, and the decline of the fever, give certain evidence of a state of convalescence. At this point some mild tonic medicine is desirable, and prepares the patient once more for the fullest enjoyment of health. This is the most successful mode of treating cases of scarlatina in its milder forms. With children, however, it is

Port. Chlor
lod Carb & 31. To be taken dissolved
in water in 24 hrs

better to trust to the soothing effects of warm poultices round the throat, than weaken the child by loss of blood.

The severe forms are characterized by the less swollen state of the tonsils, and by their being more livid and gorged with blood; by the ulcers being deeper and more spreading; and by the slough being fouler than in the milder varieties. As there is a greater tendency of parts to run into mortification, the necessity of adopting a more stimulating plan of treatment, and one more calculated to support the powers of the constitution, is manifest, and experience has shown this view of the case to be correct. The administration of wine, and of the "*extractum carnis Liebigii*," should therefore be the basis of the treatment of such cases. The quantity of wine for an adult may be from four to six ounces in twenty-four hours, and for a child about half that quantity. The wine may be either port or sherry, and should be drunk in small quantities, mixed with two-thirds water; or it may be given with sago, arrow-root, jellies, or other slops. The earlier the wine is given in the disease the better, and when delirium does or does not exist; regardless, also, as to whether the tongue is moist and white, or brown and dry; and it should be continued till the patient is decidedly convalescent. Liebig's extract of flesh should be given like beef-tea, as a drink. While pursuing this plan, it is necessary that the patient's bowels should be attended to. The treatment by wine is often extremely successful; and, as it is in general pleasant to the patient, whether a child or an adult, it is seldom refused. In cases more severe brandy may be required, or carbonate of ammonia in liquor cinchonæ, chlorinated soda, or creosote.

It may be proper, before adopting any special continuous mode of treatment, to follow the emetic first given by a dose of calomel, as a purgative, and this especially with children, to be followed in six or seven hours by castor oil or magnesia; and the bowels are ever afterwards to be kept open by remedies suited to the state of the patient and the nature of the disease. The following are the principal indications which must guide the treatment:—If there is much excitement of the system, depleting cathartics are to be given: if nausea and vomiting prevail, a seidlitz powder is of service. If the discharges from the rectum are acrid and acid, with acidity of the stomach, magnesia is preferable; if there is abdominal pain, castor oil with opium (WOOD).

Bleeding is not successful in combating affections of the larynx; on the contrary, the most beneficial mode of treatment appears to be that of moderately supporting the powers of the patient by wine and mild tonics.

Again, when the synovial membranes inflame, and the joints become enlarged and swollen, all stimuli should be withdrawn, and a moderate action of the bowels should be kept up by means of the sulphate of magnesia, with camphor mixture, or carbonate of ammonia; and if pain be severe, some sedative should be added, as the tincture of hyoscyamus in a dose of fifteen minims.

The more formidable affection in scarlatina is dropsy; and from the great tendency to effusion into the head and chest, an active treatment is necessary. We should have imagined that in dropsy, a symptom in most cases of great debility, and following a disease whose characteristic is great depression, bleeding would have been dangerous and improper; but experience has shown that bleeding by leeches over the region of the kidneys is often of service; especially if œdema appears in the face, and is accompanied by headache, some blood should be taken—from two to four ounces in the child, and from four to eight ounces in the adult. The good results of cupping are also very remarkable; and even of continuously hot poultices over the lumbar regions, when it is not thought advisable to take blood. By these means renal congestion is relieved, and the urine becomes more copious and less albuminous.

Diaphoretic doses of antimony, and moderate but not severe purging, may be had recourse to. The compound powder of jalap, or the bitartrate of potash alone in drachm doses three times a day, are among the most useful; or it may be given as an electuary, in which the cream of tartar is mixed with nearly an equal quantity of honey, treacle, or marmalade, and flavoured, if necessary, with a few drops of peppermint oil; digitalis also is much recommended, but it does not appear to possess any specific virtue. Dr. Andrew Anderson recommends the use of mercury in the form of blue pill, given twice or thrice daily, with squill and digitalis, till the urine resumes its natural appearance. The patient must at the same time be well fed; and preparations of iron may be given with advantage. The muriated tincture seems to have the best reputation; and the iodide of potassium in small doses is also useful. With this latter remedy the syrup

Equal parts Honey & Cl. & Honey
Tinct - Capivi 3i to Sy. Rose 3v

of the iodide of iron may be combined, if it is desirable to continue the *chalybeate*; or syrup of the phosphate of iron in drachm doses.

Blisters, as a means of relieving the throat, are unnecessary, and are better omitted.

Gargles are unnecessary for children, for they cannot gargle; but they are of the greatest service, especially the deodorizing gargles or washes, when the patient can be taught to use them. A weak solution of chloride of lime, or of chlorine water, or of Condry's fluid, or of the permanganate of potash, is well adapted to such a purpose. But the following is recommended as the most effectual gargle:—

Solution of peroxide of hydrogen (containing ten volumes of oxygen), six ounces; tincture of myrrh, an ounce; rose water, five ounces (RICHARDSON).

This gargle may be used at pleasure: it is refreshing to the patient, and removes the offensive secretions readily. In the case of young children, who are unable to use a gargle, the throat may be washed out, by holding the little patient with the face downwards, and by pumping the solution over the surface of the fauces through a bit of gum catheter from a double-acting india rubber bag (Richardson, *Clinical Essays*, p. 110). As an invariable routine practice, Dr. W. T. Gairdner strongly recommends that "*the patient inhale the steam of hot water from the beginning to the end of the fever; as long at least as the throat is sore.*"* In slight affections it is sufficient to employ infusion of lintseed in water, acidulated with nitro-muriatic acid, weak solutions of alum, nitre, or common salt. When membranous diphtheritic patches are observed on the fauces, and the colour of the mucous membrane is of a dark red, capsicum infusion, or powdered red pepper, is an excellent application (WOOD); and in children who cannot gargle, it may be applied with a hair pencil. Solution of zinc or nitrate of silver are also of service.

These details are given because the physician must decide, upon

* Inhalers for the purpose may be had of Mr. Young, surgical instrument maker, 58 North Bridge, Edinburgh; Mr. W. B. Hilliard, of Glasgow; and of Mr. Matthews, 9 Portugal Street, Lincoln's Inn Fields, London; and most other surgical instrument makers.

the merits of the individual case, the nature of the treatment he will adopt. But it must be remembered that cases of scarlet fever, if left to themselves, with rest and careful nursing, will generally get well. The mere intensity of the fever is no ground for active interference by way of treatment, if the pulse is full and of good strength. Much is to be trusted to the shortness of the fever, remembering that there is no disease in which the patient is more apt to be delirious, with less danger, than in scarlatina, (W. T. Gairdner, *Clinical Medicine*, l. c.)

Dietetic and Preventive Treatment.—The diet of the patient should be slops, light nutritious broths, and jellies. Fumigation will not, it should be remembered, destroy the miasmata in the sick-room; and, consequently, the doctrines of cleanliness, of ventilation, and of separation, are as imperative in this disease as in small-pox. We cannot disinfect the walls of the chamber, nor the clothes of the patient, except by washing them, or exposing them to a dry heat exceeding the boiling temperature. In general, then, the chamber where the sick patient has lain should be whitewashed and well scoured after the disease has subsided, before any person susceptible of the poison be allowed to sleep in it. It is important to guard against cold during convalescence. Children are not safe till the desquamation of the skin shall have been thoroughly accomplished—till all the old cuticle has been removed, and till the skin has resumed its natural softness. Therefore keep the patient in bed till convalescence is perfectly established; and subsequently insist on his being confined to his room, or in a room whose temperature is not below 60° Fahr., till desquamation is complete. If the whole surface of the body is well rubbed over once or twice a day with common olive oil, or a solution of glycerine in water (one part glycerine to three parts water), the irritation of the skin will be soothed, and the process of desquamation facilitated; and as soon as the child has strength to bear it, he ought to have a warm bath every second night, in which he may be well rubbed over with oatmeal and bran.

The ears of patients suffering from scarlatina ought to be carefully watched; and any complaint of pain ought at once to suggest an examination of the ears. A leech or two and warm poultices, followed by blisters if necessary, may subdue the lesions of the ears which are apt to supervene. No stimulating applica-

tions should be used, beyond the frequent syringing with warm water.

Different prophylactic medicines have been recommended; amongst which belladonna has had the greatest number of advocates; but its value has diminished greatly, as the weight of testimony is against its possessing any prophylactic virtues

(WOOD). *Side opinion of Watson & Wampler opinion of Ruffer.* Nov 18-69

HYBRID OF MEASLES AND SCARLET FEVER.

LATIN, *Rubeola*; FRENCH, —; GERMAN, *Rötheln*—Syn., *Feiërmasern*; *Ritteln*, *Falschen Masern*.

Definition.—A specific eruptive disease, preceded by, and accompanied with fever, watery discharges from the eyes and nose, sneezing, and sore throat. The eruption appears on the third or fourth day, and consists of crimson stigmata, rapidly running together into patches of an irregular shape, with obtuse angles, and of sizes varying from a threepenny to a crown piece, according to the severity of the case. The eruption continues from six to ten days, and terminates in desquamation by furfuraceous scales.

Pathology.—Those diseases now fully considered in the previous pages—namely, *small-pox*, *measles*, and *scarlet fever*—have been termed exanthematous diseases by some nosologists, in consequence of their principal phenomena being a very marked eruption.

The Arabians first described them, and considered them as varieties of one and the same disorder. Many essential differences, however, were soon observed to distinguish small-pox; but the points of resemblance between measles and scarlet fever were so many that it was not until fatal accidents had occurred, from the great error of confounding them, that their differential characters were remarked, and their separate identity established. Now it is a generally received doctrine that measles and scarlatina, in their essence and in their symptoms, present two well-defined states of disease. This is, indeed, one of the most indisputable facts in Pathology. By Schönlein, measles has been classified as a peculiar exanthematic form of catarrh; and scarlatina is placed amongst the group of erysipelatous diseases; while, according to the experience of Dr. Küttner, of Dresden, there are “Androgynous” cases calculated to embarrass the most

experienced “diagnostiker.” Measles and scarlet fever were especially confounded under the common name of *morbilli*; and even as late as the middle of the eighteenth century, writers of the highest repute supported the identity of measles and scarlet fever (the *morbilli confluentes* of Sir William Watson).

All authors before Sauvages (1768) had used the term *morbilli* (the term now in use) to designate measles; but he adopted a new name, and called measles by the designation of “rubeola”—an innovation which has caused much confusion, having been adopted by some (such as by Willan and Bateman) and rejected by others. Hildebrand, following the old nomenclature, calls measles *morbilli*, and scarlet fever *scarlatina*; and terms the disease now about to be considered *rubeola*, as has been done by Dr. Copland. The German authors call it *rötheln*, and by this name it was first described by a most distinguished and learned Scotch physician, Dr. Robert Paterson, of Leith, in 1840. He is the only physician in this country who has given an original description of the disease in the English language, his description of the disease being drawn from many cases of it which occurred in his practice.

A difference of opinion prevailed amongst authors as to whether or not this disease is of a distinct and specific form. Those who have most recently described it (Hildebrand, Paterson, and Copland) consider it to be a disease possessing characters common to both measles and scarlet fever, as well as characters peculiarly its own. In truth, it seems to be a hybrid disease, developed from combined poisons of the two fevers, measles and scarlet fever. Dr. Küttner, of Dresden, states that he has seen occasionally in the same individual portions of the skin presenting the scarlatina eruption, while in other parts the eruption of measles was to be seen. He thus recognized not only examples of transition, but he recognized cases which may be termed hybridous (*Dublin Hosp. Gazette*, 15th Dec., 1858; and Ranking's *Abstract*, vol. xxix., p. 20).

Symptoms.—The febrile stage of the disease varies, like all the diseases already noticed, not only in the severity of the symptoms, but also in the length of the attack when compared with scarlet fever. It usually commences with rigors, not severe, but continuous. More or less cough soon makes its appearance—of the same clanging nature which is observed in the febrile stage of

true measles—and is very shortly accompanied with itchiness, redness, and weakness of the eyes, lachrymation, frequent sneezing, and watery discharge from the nose. In persons more advanced in life, severe frontal headache is complained of, together with rheumatic pains, more especially in the muscles of the back and chest, nausea, and sometimes vomiting, together with constant drowsiness. The skin is hot and dry, with the pulse above the natural standard.

A greater or less number of these symptoms is always noticed; but, in addition, sore throat is a most constant one. This, in some cases, is extremely slight, amounting only to a roughness of speech and trifling difficulty in swallowing; but in others it goes on to severe inflammation of the *tonsils, velum pendulum palati*, and surrounding parts. This last inflammatory affection is, however, more severe during the eruptive stage. The sore throat is one of the most characteristic features of the disease, occurring in the slightest and most gentle cases (ROBERT PATERSON). MB

The odour given forth by patients under this disease is described by Dr. Heim, of Berlin, as similar to, but stronger than that which scarlatina patients emit, and has been likened to the smell of a place where fish is kept—in short, fishy.

When the febrile state now described has continued for three or four days, the appearance of an eruption is sudden and general. It breaks out all at once over the whole body, and consists of bright and thickly set stigmata which appear on the trunk, but are more sparingly dispersed over the face and extremities. It assumes different aspects and degrees of confluence, according to the severity of the case. Its first appearance resembles measles, but the stigmata rapidly run together, and soon assume an irregular shape, with obtuse blunt angles. These irregular patches are of an intense red colour towards the centre, being gradually shaded off towards the margins, which approach in colour that of the surrounding skin. The size of the patches in ordinary cases seldom exceeds a sixpenny piece; but in the severe forms of the disease they run still further together, and are to be seen of the size of a crown piece. In such cases, which are usually of a malignant nature, the whole body may be covered over with patches, varying from the size of a sixpenny piece to a crown piece, thickly set together, and of an intensely dark colour towards their centres. The eruptive patches are felt to be distinctly elevated above the

skin—some more than others, and always greatest in the centre of the patch.

During the continuance of the eruption, the general symptoms already described are usually aggravated, and not unfrequently new symptoms are superadded. The sore throat becomes much worse. The hoarseness becomes so great as frequently to cause entire loss of voice, and generally more or less external tumefaction of the throat takes place. In severe cases this is great, and is accompanied with much redness and swelling of the throat internally. There is a total inability to swallow even the slightest portion of fluid, which generally regurgitates by the nose. A large secretion of mucus of a vitiated nature takes place, the cough is constant, and is rendered doubly severe by the state of the throat. The pulse is very frequent; the skin hot and dry; and there is great restlessness, expressed by children tossing the head frequently from side to side, accompanied with frequent starting; and they are sometimes seized with convulsions. It is in this stage, in the worst forms of the disease, that death generally occurs, and that by coma. It may, however, take place either by suffocation from the large quantity of vitiated mucus, or by convulsions and subsequent coma. Vomiting is an occasional symptom during this stage, and, like convulsions, is sometimes seen in mild cases of the disease in children.

The eruption in mild cases, in general, continues distinct for from four to five days, during which time the other symptoms are going on favourably, becoming gradually milder as the period of the decline of the eruption draws near. In severe cases, however, the rash keeps its bright colour and distinct form for a much longer period—*e. g.*, six, eight, or ten days.

The termination of the eruptive stage is, in some instances, marked by what is termed a distinct crisis,—such as the occurrence of copious sweating, deposits from the urine, diarrhoea, and epistaxis. Most commonly, however, there is no such crisis, but the eruption gradually fades, and the disease subsides.

As this happens, the desquamation by furfuraceous scales gradually ensues. This event is indicated by the appearance of scales towards the centre of the patches of eruption, to the margins of which they gradually extend, and soon spread over the whole body. The scales are small, and not unlike those of measles. On the hands and feet the scales are larger, but

never reach the size of those of scarlet fever (ROBERT PATERSON).

Lesions Seen in Fatal Cases.—The accounts of these are few in number. They vary according to the period of the disease at which death occurs. Death most frequently happens during the eruptive stage, from coma, or from the affection of the throat and lungs. No morbid appearances of a uniform nature can be observed connected with the mode of death by coma; but when death happens from pulmonary oppression, the lungs are found much congested, the mucous membrane of the bronchia injected, with a copious mucous secretion. The throat presents very similar appearances to those which are seen in scarlatina,—great tumefaction, and dark coloration of the membrane lining the throat, dark aphthous spots, and large quantities of vitiated viscid mucus.

Diagnosis.—The accompanying febrile symptoms at once distinguish the disease from roseola, as also do the peculiar characters of the eruption. The only other affections with which it may be confounded are measles and scarlet fever. The following table points out the diagnostic marks more clearly by contrast than can otherwise be done, and shows that rubeola, rötheln, or the mixed disease, has every right to be considered as a distinct affection:—

TABLE SHOWING THE MOST PROMINENT DISTINGUISHING CHARACTERS OF SCARLET FEVER, RUBEOLA, AND MEASLES (Paterson).

SCARLET FEVER.

RUBEOLA OR RÖTHELN.

MEASLES.

Symptoms of First Stage, or Premonitory Fever.

Rigors; nausea; sometimes vomiting, thirst, and heat of skin; with sore throat, hoarseness, and delirium, in the anginose variety of scarlatina.

Shiverings; nausea; rarely vomiting; itching; redness and pain of the eyes, with increased flow of tears; sneezing, and watery discharge from nose; cough, sore throat, and hoarseness.

Rigors; nausea, and sometimes vomiting; frequent starting during sleep; itching; redness; pain of eyes; watery discharge from eyes and nose; sneezing; harsh cough.

Duration of Premonitory Fever.

Premonitory fever is of short duration; the eruption most generally making its appearance on the second day.

The eruption generally breaks out on the third or fourth day, so that the premonitory fever is prolonged over that time.

Eruption makes its appearance towards the close of the third, or beginning of the fourth day.

SCARLET FEVER.

RUBEOLA OR RÖTHELN.

MEASLES.

Appearance of Exanthematous Eruption.

It first appears in innumerable red dots or points, being at first of a pale red colour, soon acquiring a deeper tint, and at last giving the affected portion of skin a uniform red appearance.

The rash appears in minute dots, and rapidly assumes the appearance of irregular-shaped patches, with obtuse angles, varying in size from that of a three-penny to much larger than a crown piece. The red rash is gradually shaded off with the surrounding skin.

The rash appears in minute red points, like flea-bites; several of them soon coalesce, and form rounded masses, irregular-shaped crescents, or semicircular patches.

Roughness or Elevation of the Affected Skin.

There is a perceptible roughness in the skin affected with scarlatina. It is in general most evident on the breast and extremities, and seems to consist of the enlarged papillæ of the skin.

In this disease, more especially in the severer forms of it, the patches of eruption are distinctly and considerably elevated, and more especially towards the centre of the patch.

The elevation of the patches of eruption in measles is slight; though in general distinct in the worst cases, they are certainly not at all elevated as a rule.

Part of the Body First Affected.

The efflorescence is first perceptible on the face, neck, and chest, gradually passing downwards, and becoming diffused over the whole body.

The efflorescence first appears on the trunk of the body, the whole of which it at once occupies. It is always more sparingly seen on the extremities, but seems to break out there at the same time as it does on the trunk.

The efflorescence first appears on the forehead and among the roots of the hair, and spreads slowly and successively over the neck, chest, trunk, and extremities.

Duration of the Eruption.

The eruption remains present three days; begins to disappear on the fourth day; and is almost entirely gone by the termination of the fifth day.

In the rötheln the duration of the eruption seems to depend upon the severity of the disease; in mild cases remaining out four or five days, and in bad cases six or ten days.

In this disease it remains out three days.

Symptoms accompanying the Eruption.

The symptoms which accompany the eruption in each of the three diseases are quite the same as those of the premonitory fever. It is proper here, however, to remark, that it is only in the anginose and malignant varieties of scarlatina that we have sore throat, there being little or none in the simple scarlatina, while in the mildest kind of rötheln this is always a prominent and troublesome symptom.

SCARLET FEVER.

RUBEOLA OR RÖTHELN.

MEASLES.

Desquamation.

The cuticle in this disease is thrown off in patches of considerable size, the largest being from the hands and feet.

The desquamation of rötheln consists of minute portions of cuticle, like scales of fine bran.

The desquamation always begins towards the centre of the eruptive patch, and gradually extends to the circumference.

The desquamation of measles consists of minute portions of cuticle, like scales of fine bran.

Sequelæ.

Anasarca is the most common sequela of scarlet fever. It is extremely common, and most frequently occurs after the mildest cases; swelling and suppuration of the cervical glands is also common.

"I have noticed one case of dropsy after a mild, though well-marked attack of this disease; swelling and suppuration of the cervical glands also frequently takes place" (DR. PATERSON).

Affections of the lungs and pleura; tedious distressing cough; chronic bronchitis; pneumonia, tubercles; gangrenous inflammation of cheeks, gums, lips, genital organs, &c.; dropsy occasionally occurs, but very rarely; diarrhoea is very common after some epidemics.

Prognosis.—It requires to be as guarded as in scarlatina; for, like scarlatina, rubeola is often an extremely and rapidly fatal disorder. The greater or less acuteness of the premonitory fever generally affords us a means of judging as to the probable severity of the eruptive stage; and in general it is a mild disease. To have a copious secretion of mucus in the back of the throat is always a bad symptom, or regurgitation of fluids by the nose. The chest ought to be examined from day to day, as sudden inflammatory action is apt to be established, and often it rapidly proves fatal. The condition of the urine requires also to be daily investigated.

Treatment.—The treatment is similar to that of scarlet fever. The functions of the skin are if possible to be stimulated; and Dr. Paterson found that the *aqua acetatis ammoniacæ*, in the proportion of two ounces to half an ounce of antimonial wine and four ounces of water, made into a mixture, was the most useful agent. The use of colchicum was also had recourse to with decided benefit.

DENGUE.

LATIN, *Denguis*; FRENCH, *Dengue*; GERMAN, —; ITALIAN, —.

Definition.—A peculiar febrile disease, commencing very suddenly, and conjoined with severe pains in the joints, which swell; succeeded by general heat of skin, intense pain in the head and eyeballs, and the appearance of a cutaneous eruption or efflorescence on the third or fourth day, commencing on the palms of the hands and spreading rapidly over the whole body. It rarely continues visible beyond twenty-four hours. Remissions and relapses are numerous; and the disease may persist about two months, marked by great prostration and cachexia. The relapses are marked by rheumatic or neuralgic phenomena. It occurs in the West Indies and elsewhere.

Pathology.—This disease seems to combine an exanthematous eruption ushered in by fever, with a rheumatic or neuralgic state; and the course of the malady is so divided by intervals and remissions as to give one the idea that relapses are of frequent occurrence in its course. It has been chiefly prevalent in Rangoon, Calcutta, Berhampore, Patna, Benares, and Chunaighur, in the East Indies; the Island of St. Thomas in the West Indies; the Southern States of America; the ports on the Gulf of Mexico; the towns of New Orleans, Savannah, Charleston, Philadelphia, and New York. It was epidemic in America in 1824-28; and nothing appears to have been heard of it again till 1847 and 1850, when it again visited the Southern States. An epidemic of dengue has been recently described by Lemmon as having occurred in Virginia (*Amer. Med. Times*, Feb. 16, 1861). It is not known as an epidemic disease in Great Britain. It has been described by Nicholson, Mellis, Kennedy, Wood, Cavell, Twining, Mouat, and Goodeve. Dr. Richardson, in his admirable *Clinical Essays*, notices the circumstance that we not unfrequently meet with "scarlet fever connected with acute rheumatic fever;" and he ascribes the first notice of this connection in this country to Dr. Golding Bird. Subsequently the circumstance was noticed by Dr. Kelso, of Lisburn, and by Dr. Ross. Dr. Andrew Anderson, in his *Lectures Introductory to the Study of Fever*, notices that rheumatic pains of the arms and legs, often very severe, connected, as he supposes, with the poisoned state of the blood, are

not unfrequently met with. Lastly, Dr. Richardson himself records the cases of four children, in which the rheumatic state combined with scarlatina was distinctly expressed (*Clinical Essays*, p. 85); and Dr. Wilkes has noticed similar cases.

Symptoms.—The invasion is very sudden, and the development rapid. In the greater number of cases the first symptoms have been headache, with intolerance of light, restlessness, and more or less chilliness, debility, pains in the back, the limbs, and joints. The small joints swell, and there is soreness, with stiffness of the muscles. The skin soon becomes hot and dry, the pulse frequent, the face flushed, and the eyes red and watery. The tongue, though red, is usually clean. A rash or papular eruption sometimes appears, though not generally at this stage. Painful swellings in the lymphatic glands of the neck, axilla, and groins are common. The testicles also swell, and continue so till the subsidence of the other symptoms. The febrile state lasts from twelve hours to three or four days, after which it subsides, leaving the patient very feeble. This remission lasts for two, three, or four days, when a return of the fever and pains, with a thickly coated tongue, nausea, and epigastric tenderness mark another phase of the disease. On the fifth, sixth, or seventh day the eruption appears in the form of a scarlet efflorescence on the palms of the hands, which spreads rapidly over the body, and gives relief to the symptoms of febrile irritation. The eruption is extremely variable in character, being sometimes smooth, red, and continuous, as in scarlet fever; sometimes in patches, rough, and of a dark hue, as in measles; and occasionally either papular, vesicular, pustular, or furunculous; often with a mixture of two or more of these forms. The complaint gradually subsides, and leaves the patient with some rheumatic stiffness or soreness for a longer or shorter period, with feelings of weakness and mental depression. The duration of the affection varies with the length of the remission; but on the average is about eight days. Decided implication of the mucous membrane of the mouth and throat prevailed in the last epidemic in Calcutta, with an almost entire absence of the articular pains.

Treatment.—Emetics and purgation subdue the fever; but as the disease runs a specific course, time is an essential element in the treatment. Calumba, rhubarb, and soda form a useful alterative medicine. Ophthalmia is sometimes consequent on this

disease, and is to be subdued by leeches to the inner membrane of the eyelids. Palliation and alleviation of symptoms, as they arise, chiefly by opium and alkaline remedies; following the indications given under “Scarlatina” and “Rheumatism.”

ERYSIPELAS.

LATIN, *Erysipelas*; FRENCH, *Erysipèle*; GERMAN, *Erysipelas*—
Syn., *Rothlauf*; ITALIAN, *Risipola*.

Definition.—*A febrile disease, associated with a peculiar eruption of the skin. The inflammation which attends this eruption is apt to spread indefinitely, and may involve the areolar tissue beneath the skin.*

Pathology.—As in other diseases of the miasmatic order, it is believed that in erysipelas a specific poison is absorbed and infects the blood, and that after a given period of latency it produces fever. The specific action of the poison, however, is mainly made manifest by inflammation of the skin and subcutaneous areolar tissue, which runs a definite course, and sometimes terminates in inflammation of the membranes of the brain. The inflammation and the fever are of a peculiar nature, not yet clearly understood. In Scotland the disease is known by the name of the *Rose*; in England it is sometimes called *St. Anthony's fire*.

Idiopathic erysipelas is very constantly preceded by fever—eighteen times out of twenty—and although it may be supposed that the fever is consecutive to the inflammation of the skin, yet, before the redness of the skin is seen, the temperature, if measured by a thermometer, will be found above 98·6° or 99° Fahr., and attended with general malaise. The affection of the areolar tissue may be trifling, but it is seldom altogether wanting.

The pathological phenomena which result from the action of the poison on the skin are, first, that the cutis is diffusely inflamed, the affected part being either of a bright scarlet or a rose-coloured tint, evanescent on pressure, but returning on that pressure being removed. This inflammation is usually of great extent, occupying very commonly the whole face, head, and neck, or a considerable portion of the trunk, or one or both lower or upper extremities. It runs a course which may be characterized as “tolerably regular and definite.”

It may terminate by resolution, by vesication, or by gangrene.

When it terminates by resolution, the rose tint gradually changes to a deeper and more venous hue, and at length fades away, leaving the skin of its natural colour, but with the texture so impaired that desquamation follows. If the inflammation terminates in vesication, the cuticle is raised into a number of vesicles of greater or less size, and sometimes into large bullæ or bladders containing a yellowish transparent serum. The cuticle at length ruptures, the fluid is discharged, and a crust sometimes forms, which, on falling off, leaves the skin underneath either sound or superficially ulcerated. Should the termination be by gangrene, the skin becomes livid or black, its whole texture more or less disorganized, while the bullæ or phlyctenæ which often form in these cases are filled with a bloody serum. The cutis, when examined after death, whatever may have been the form of the disease, is always found greatly thickened and infiltrated, but the redness, except in cases of gangrene, has entirely disappeared. (1) (2) (3)

It is seldom that erysipelas is limited to a simple affection of the skin. More commonly, at some period of the disease, the areolar tissue beneath the affected skin becomes the seat of a serous exudation; and it may suppurate, or proceed to gangrene. When the termination is by effusion of serum, the quantity of fluid effused is generally so considerable that the head, face, or limb, is greatly, and sometimes even hideously, swollen; and if the part be now incised, the vessels are seen enlarged and numerous, and the cellular tissue loaded with serum, sometimes turbid and flaky. The tissue is more easily torn than usual. This inflammation may terminate by absorption of the serum; but in a few cases ulceration follows, and in some gangrene. Adhesive inflammation seldom takes place in erysipelas without its being accompanied by a serous effusion and the occurrence of suppuration. Suppurative inflammation is, indeed, uniformly preceded by serous effusion, and the result may be the formation of an abscess; or, what is much more common, pus may be *infiltrated* through the areolar tissue, uncircumscribed by an adhesive inflammation—a circumstance improperly considered by some as pathognomonic of erysipelas. The parts more usually the seat of phlegmonous circumscribed abscess are the eyelids and the integuments covering the cheek-bones, and the pus in these cases is usually of a laudable and healthy character. In all other parts of the body the abscess is generally diffuse; and, the inflammation being of a low type, the pus is poor, and often little more than a (MB)

fetid sanies. Should the parts slough, the purulent fluid becomes loaded with a dirty broken-down areolar tissue, generally mixed with some loose lymph. In some instances the suppurative process extends between the muscles, causing extensive and often irreparable mischief. In the event of this inflammation terminating by gangrene, the integuments of an entire limb are sometimes detached, laying bare the muscles, a large artery, or a bone, involving the aponeuroses and tendons, and sometimes destroying the interior of a joint. Gangrene, however, does not equally take place in all parts, for it is seldom seen on the scalp, the face, or the trunk. It is the extremities, then, and more especially the leg and thigh, and also the labia and scrotum, that are apt to suffer from this affection. *part. little vascular*

The appearances found after death from erysipelas are similar in many respects to those found in cases of typhus fever.

Symptoms.—The symptoms of erysipelas arise out of the fever and local affection, and appear of various degrees of intensity.

In acute sthenic cases the erysipelatous inflammation is preceded and accompanied by fever; and the attack may be sudden, or ushered in by rigors, irregular flushings, muscular pains, accelerated pulse, white tongue, nausea, vomiting, and deranged bowels. Sore throat is an early and constant accompaniment. These symptoms, when they do exist, last for some hours—perhaps till the end of the second night or beginning of the third day—when the fever becomes continued, the tongue brown and dry, and shortly afterwards the cutaneous inflammation appears, but without any remission of the fever. The inflammation generally appears at the seat of any injury to the skin, such as a wound, and is most intense there. By some, indeed (Trousseau, for example), it is held that erysipelas always originates from some external injury or irritation, which may be very slight. But this character erysipelas has only in common with other eruptive diseases, as Mr. Paget has described in his admirable Address on Surgery, delivered to the British Medical Association at their meeting in London, in August, 1862. He noticed that, “having cut a boy for stone, the boy became very ill three days afterwards, and seemed in danger of his life; but soon a vivid red eruption appeared at and about the wound. This was measles, earliest and most intense at the seat of injury, just as erysipelas might have been. Thence it extended, and ran its ordinary course, and did no harm.” Mr

*Erysipelas distorted by liability to short relapse
& by the great varies in temper wh may occur
in the short periods of time.*

DIAGNOSIS OF ERYSIPELAS.

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I maybe assocd with Acute Rhe case Oct 20 Woman 79

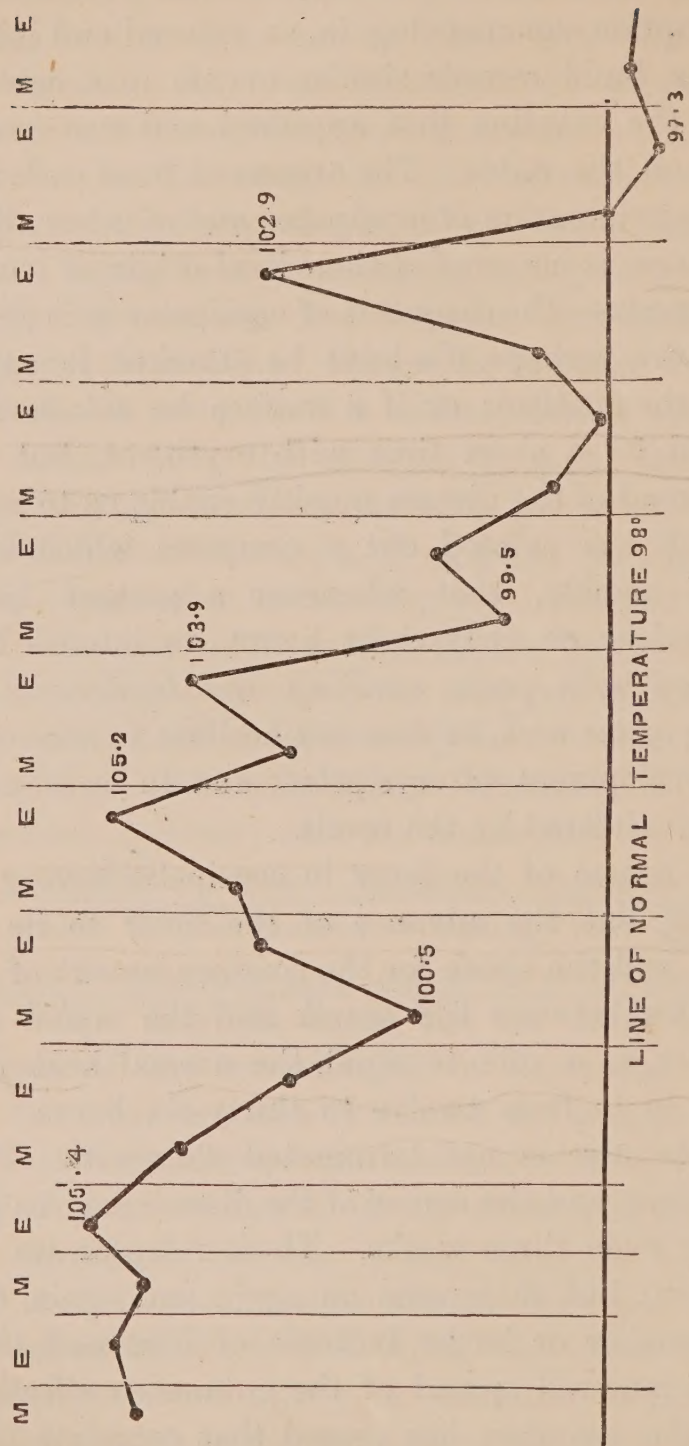
Paget states that he has seen similar events with scarlet fever, the eruption commencing in an injured and inflamed knee. Dr. William Budd records similar events in a case of small-pox, in which the eruption first appeared and was most intense over a bruise on the nates. The argument from such facts is, "that the local determination of erysipelas, and of other allied diseases, after operations, is no proof of their local origin or local nature."

Diagnosis.—The diagnosis of erysipelas is in general easy. For a few hours, perhaps, if a joint be attacked, it may be mistaken for acute rheumatism; or if a surface be attacked, it may be confounded for a short time with erythema, but the intumescence and spread of the disease quickly enable us to rectify the error.

Frank has pointed out a symptom which he considers diagnostic—namely, that whenever a patient has exhibited, for twenty-four or forty-eight hours, an intense febrile movement, attended with pain, swelling and tenderness of the lymphatic glands of the neck, he does not hesitate to announce the approaching development of erysipelas; and in no case has the diagnosis been invalidated by the result.

The course of the fever in erysipelas is very similar to that of measles; but the advance of the fever to its height continues longer, and the epoch for the commencement of the defervescence vacillates between the *fourth* and the *eighth* days. The defervescence, as a rule, is rapid, the normal heat being attained, or nearly so, in from twelve to thirty-six hours. Frequently, however, the case is not terminated therewith. New relapses may take place, and the course of the disease may be prolonged through two or even three weeks. These relapses are severally of short duration; but they come on again and again, and are ushered in by a smaller or larger increase of heat, and they are connected with a renewed spread of the cutaneous affection; and it is only after the eruption has ceased that complete and definite defervescence ensues. Very sudden changes of temperature are characteristic—4° or 5° Fahr. in twenty-four hours, or a fall of 7° or 8°, commencing immediately on the appearance of the characteristic redness (COMPTON). This erratic and protracted form of erysipelas is most frequently met with in the aged, associated with gouty or rheumatic states of the system, as well as with albuminuria or renal disease. The *local* symptoms vary according to the part affected, the mode of termination of the inflammation, and also according to the character and duration of the fever.

THE FOLLOWING DIAGRAM REPRESENTS THE TYPICAL RANGES OF TEMPERATURE IN A CASE OF ERYSIPELAS WITH RELAPSES AFFECTING THE FACE. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS.



When erysipelatous inflammation affects the face, it may begin either in the skin or in the subjacent areolar tissue. If the areolar tissue be primarily affected, the face at the inflamed part becomes swollen, but the skin suffers no discoloration for some hours, so that it is impossible to distinguish it from an ordinary attack of swelled face. At length, however, the skin inflames, and the part is now red, hot, and painful, as well as swollen, and the disease is fully formed. At the commencement of erysipelas of the face the attack is usually partial, and perhaps limited to the bridge of the

I have seen several cases of infection of the
tympanum at Paris from Erysip. 1820

nose, to one ear, to the lower eyelids, or to one cheek; but in severe cases it gradually extends, often involving the whole of the integuments of the face, head, and neck; so that at the end of three or four days those parts present a strangely swollen, disfigured, and even in some instances, hideous appearance, scarcely a feature being discernible. The nostril, moreover, is imperforate from internal swelling, so that the patient is obliged to breathe with his mouth open, while the inflammation may extend to the auditory passages, and render him completely deaf. Extension of the inflammation to the membranes of the brain sometimes takes place, while the external inflammation continues. This untoward event is followed by delirium and coma. But delirium frequently supervenes in the course of erysipelatous attacks, independently of any metastasis or extension of the disease to the membranes of the brain. It commences with wandering of the mind at night, similar to that which is observed in fever. Utterance is given for the most part to low, muttering, and rambling expressions, which rarely assume a noisy character, but which in fatal cases terminate by coma. When the patient has been of dissipated habits, or is otherwise of a dilapidated constitution, then the delirium resembles that of *delirium tremens*, not due to inflammation of the brain, but in consequence of an altered condition of the blood and of the nervous system (BARCLAY).

Brain

On the fourth, sixth, eighth, or some later day, the bright red colour of the skin changes to a deeper hue; the serum effused is absorbed, desquamation takes place, and the skin gradually returns to its natural colour. It is not unusual, however, for abscesses to form, particularly on the eyelids or cheeks, and which, being opened quickly, heal, and hardly retard the convalescence of the patient. In some cases the disease becomes erratic, and extends over the chest or down the back, and desquamation is seen going on in one part while the erysipelas is spreading in another. This is characteristic of cases associated with gouty or rheumatic constitutions.

Abscesses

The extremities are more commonly the seat of erysipelatous inflammation than the trunk, and the lower extremities are more frequently affected than the upper. When these parts are affected, the fever is less severe than in erysipelas of the head; but the local symptoms are generally more formidable, for the degree of heat is greater, and the pain so severe that the weight of a sheet can hardly be borne. The inflammation likewise

often involves the lymphatic vessels, when they can be traced by white or red lines for many inches, as from the knee or elbow to the inguinal or axillary glands; and these sometimes enlarge and suppurate. If the erysipelatous inflammation ends in suppuration, the abscess is always diffuse, and the swollen limb gives a peculiar sensation to the hand; and which has been compared to what a person feels with his feet on passing over a quagmire. The dark, black, discoloured appearances of gangrene are too obvious to render any description of the parts so affected necessary. Numerous varieties of erysipelas are referred to in practical works, especially surgical, most of which are modifications of the disease as above described.

Besides the *erratic* form just noticed, there is the—

Phlegmonous Erysipelas (Erysipelas Phlegmonodes) and *diffuse inflammation of the cellular tissue*, in which the inflammation extends deeply into the subcutaneous tissues, and tends to spread indefinitely. It is attended with greater pain and swelling than the more superficial variety, and usually the general symptoms are more severe. Suppuration and gangrene of the areolar tissue are not uncommon; and if the disease penetrates beneath the fascia, the sufferings of the patient are greatly aggravated by the compression of the inflamed parts, and much organic mischief may result from the confinement of pus and the various products of the gangrenous state.

Gangrenous Erysipelas (Erysipelas Gangrenosum).—As the name implies, this form is accompanied with death of parts, and the tendency to death of tissue may be due either to the inherent depressing nature of the disease, or the depraved state of the system, as of the blood, the co-operating influences of an epidemic constitution, debility, confined and impure air, as in crowded hospitals, unwholesome or scanty food, or simply the excessive violence of the inflammations. The peculiar hot and burning pain, with the purple or livid hue of the redness, indicate the tendency to gangrene; and its near approach is shown by the slowness with which the blood returns after removal by pressure, and by the formation of vesicles (phlyctenæ) filled with turbid reddish serum. These vesicles are to be distinguished from those which are to be seen on the skin in severe contusions on fractured limbs. The fluid in the vesicles of gangrene can be pressed from under one part of the cuticle to another, which is not the case in the vesicles on a fractured limb or a severe

bruise. Patients with typhoid fever, infants soon after birth, and young children, are most frequently the subjects of gangrenous erysipelas; and it is not uncommon in hospitals, during the prevalence especially of malignant epidemics of erysipelas.

Cause.—The mystery which hangs over the origin of disease poisons does so, in a remarkable degree, over erysipelas; for this disease is often epidemic, and appears to be very constantly present in communities, and especially in large towns.

The predisposing conditions are age, mechanical or chemical injuries, as blows, punctured wounds, and incised wounds generally, bites of insects, or burns; also certain articles of diet, as mussels or periwinkles; and many diseases likewise, as dropsy with renal disease, typhus fever, and others of a debilitating kind. The effects of age in predisposing to this disease are considerable. New-born children, for instance, are occasionally subject to it, but from that period to adult age it is seldom witnessed. The period of life most subject to acute attacks is from twenty to forty; and to frequent asthenic attacks from forty to old age. Both sexes suffer in nearly equal proportions.

Propagation of the Disease.—The spread of erysipelas has been so frequently observed, both in the sick-room and in the wards of hospitals, that no doubt can exist of this disease being communicable by impalpable emanations. In the year 1760 erysipelas spread so extensively through the wards of St. Thomas's Hospital, in London, that it was believed the plague was in the hospital. Dr. Baillie described it as spreading also in St. George's Hospital, London; and Dr. Cullen, in the Hospital at Edinburgh. It has been found to spread extensively on board ship; and Drs. Wells, Watson, and others, have given several remarkable instances of its spreading in families. Dr. Steele writes, in his excellent *Annual Report on Guy's Hospital* for 1863, that "for some years past it has been customary to place patients suffering from erysipelalous wounds in these (the medical) wards, in order to diminish as much as possible the risk of extending infection in surgical wards, as well as to promote recovery in the patients themselves. It happened, however, that in one of the wards of the new hospital into which a patient suffering from erysipelas was placed in the course of the past year, *five* persons suffering from other complaints were attacked with the disease; and although none of the cases were attended with fatal consequences, the occurrence is sufficient to point out the danger which must

be occasionally apprehended." Dr. Maclachlan, on the other hand, has never seen the disease propagated by contagion or infection in the infirmary of Chelsea Hospital; and he is disposed to think that the disease is less contagious or infectious when occurring in persons of advanced life than at other periods.

That it is communicable by some palpable virus, was shown by Dr. Willan, who says, that if a person be inoculated with the fluid contained in the phlyctenæ or vesicles of a genuine erysipelas, a red, painful, diffused swelling and inflammation analogous to erysipelas is produced. The danger, however, attending this experiment has not allowed it to be repeated.

Erysipelas also spreads by *fomites*. In hospitals, wards are occasionally obliged to be cleared out, to stop the continued spread of this disease. In the navy the spread by *fomites* is so well understood that it is debated whether swabbing the decks or dry rubbing them is the best mode of disinfecting a ship, and preventing the spread of the disease. It has spread extensively, and for long periods, in the Birmingham, Edinburgh, Glasgow, and London hospitals, and is only got rid of by emptying and whitewashing the wards. It is said, however, that dry rubbing is preferable to washing, moisture appearing to promote the extension of the disease. The old "Dreadnought" hospital ship in the Thames was so impregnated with the *fomites* of erysipelas that she had ultimately to be broken up, and a new vessel substituted.

A patient having passed through an attack of erysipelas has no security against future attacks of the disease; and many persons suffer repeatedly from erysipelas; some periodically. There appears to be a constitutional predisposition to the disease in some people, and especially in those who have periodic attacks. Some women have attacks every month. Intemperance, and all influences which tend to depress the system, predispose to the disease, and hence partly the prevalence of the disease in hospitals. But there are unquestionably some unknown conditions of the atmosphere which seem to favour the dissemination of the disease. It has been observed that this predisposition to erysipelas exists in the ordinary wards of hospitals at the same time that puerperal fever prevails; and it was formerly not an unfrequent accompaniment of small-pox.

Period of Latency.—Erysipelas has occasionally followed a few hours after exposure to the infection. Dr. Elliotson thinks five

2-14 days probable incubation

days elapsed in his own case, and Dr. Watson has given three cases in which the interval was a week. It has been observed in hospitals that a fortnight has elapsed after its subsiding in one case and appearing in another in the same ward. It is probable, therefore, that the period varies from two to fourteen days.

Erysipelas and puerperal fever are interchangeable diseases, the one being able to induce the other by personal contact. Destructive epidemics of erysipelas have now and then occurred in Europe, and several parts of America have of late years been the scene of similar ravages, especially in the New England States, the South-Western States, and the interior of Pennsylvania (DR. WOOD).

Prognosis.—The most experienced physicians consider erysipelas, at all periods of life, “a dangerous and deceitful disease;” and when it fixes on the face or scalp it is one of the most serious diseases to which an aged person especially is liable; for when all appears to be going on well, the membranes of the brain may become involved, or the powers of life may give way, the patient sinking suddenly under the depressing influence of the poison. The disease may extend to the fauces or the glottis, and the patient then dies suddenly from the œdema of the glottis which supervenes. The disease is peculiarly fatal to drunkards and to patients of broken-down habits; and frequently recurring attacks show such a bad state of health as indicates a speedy break-up of the constitution (MACLACHLAN). It is five times more fatal to people above sixty than to people between fifteen and sixty years of age (*Registrar-General's Fifth Report*, p. 456).

Treatment.—Broussais states that when he served with the French armies in Italy, he has seen erysipelas allowed to run its natural course, and the result was, that it made immensely rapid progress, and ended either in suppuration, in gangrene, or in fatal visceral inflammation.

Erysipelas, in the opinion of some, is a disease of simple inflammation, and consequently ought to be treated by general and local bleeding; while, on the contrary, others contend that it is a specific inflammation; and long experience has shown that bleeding is often injurious, while a tonic mode of treatment is much more uniformly successful.

There are very few physicians, from the days of Hippocrates to the present time, who have not bled patients in erysipelas, and consequently this experiment has been made on a large scale;

still, many of the warmest advocates of bleeding admit that the operation is occasionally followed by unpleasant consequences. Indeed, the treatment by bleeding has been often followed by so many unfavourable results, that many physicians, the most intelligent of the profession, affirm that, according to their experience the practice is not only unfavourable but highly injurious. Andral is reported to have said, "In erysipelas with delirium, bleeding pales the skin, but the disease continues; the cellular tissue remains gorged, and death follows. We open the body, but find nothing." Cruveilhier says, "*des erysipèles rentrés*" is a consequence of unusual or too abundant bleeding, and he considers the question of bleeding in this disease to have been "*depuis longtemps jugée*." Blache and Chomel likewise say that "Experience has proved that general bleeding has no other effect than to blanch the eruption, without notably abridging its duration." In this country, Drs. Fordyce, Wells, Pearson, Heberden, and Willan, all give their testimony to the frequent ill effects of bleeding in this disease; and, in consequence, they, for the most part, recommend a tonic treatment.

It is therefore to be recollected that bleeding will not cure the *erysipelalous* inflammation, in the way that it produces a salutary effect on an idiopathic inflammation of the lungs, occurring in an otherwise healthy person. It is also to be borne in mind that as a rule, bleeding is not borne well by persons suffering from erysipelas; and it is necessary to be ever mindful of the fact that people of a certain class in populous towns cannot bear bleeding so well as those who pass their life in the country. For instance, a brewer's drayman in London, accustomed to rejoice in the beverage which he delivers to his customers, would sink suddenly under the influence of a bleeding; when if double or even treble the amount of blood were abstracted from a countryman suffering from erysipelas of a sthenic form, but heretofore in good health, it would produce but little effect, and that probably for good. Bleeding, as a rule, is only indicated in the young, the healthy, and the vigorous; and it must equally be avoided entirely in the aged and in broken-down cachectic patients.

Rest, saline laxatives, cooling drinks, and low diet, are the elements of treatment in mild and simple cases. An emetic is useful at the commencement; and I have seen, in the practice of an eminent surgeon, that frequent resolution of an erysipelalous attack has followed an antacid laxative, such as of rhubarb

powders and carbonate of soda, together with the counter irritation of a mustard poultice over the stomach. Laxative and cathartic remedies are to be selected and apportioned according to the violence of the attack and its nature, as tending to the unfavourable results of the specific inflammation already noticed. Calomel is a most valuable purgative, as a sedative in febrile disturbance, especially when followed by castor oil, or the common black draught. The indications to the use of certain remedies, as given in the treatment of scarlet fever, are equally applicable here. gr v

If the febrile state is not subdued, antimonials are of great service, for so far as they are diaphoretic in their action they tend to subdue the vascular excitement. If symptoms of nervous depression ensue, opium, or opium and ipecacuanha are indicated, also wine and quinine, ammonia and camphor, in asthenic cases with a tendency to a typhoid state. The *tincture of the perchloride of iron*, in doses of ten to thirty minims, *three, four, or five* times a day, in water, infusion of quassia, or calumba, is now also a remedy much in use, and it may be alternated with the *syrup of the phosphate of iron*, in doses of a tea-spoonful three or four times a day. In cases where the system is obviously gouty or rheumatic, and where the joints are affected, colchicum with saline diaphoretics are the most efficient remedial agents. Oleo Ant. for vi-vii

Local applications are potent for good or evil, and must therefore be used with great caution. The effects seen on the skin do not constitute the whole disease; and if the development of these processes on the cutaneous tissue is imprudently interfered with, there is imminent danger to internal organs. To check the advance and prevent the encroachment upon new territory, rather than to subdue it if already in possession, ought to be the sole aim of local treatment; and to mitigate the local pain and uneasiness. Bland mucilage, such as that of viscid lintseed tea, from which light muslin cloths have been steeped and spread over the inflamed surface, sometimes affords relief. Dry flour, or rye-meal, frequently dusted from a dredge-box over the erysipelatous patches, are soothing applications. A lotion of nitrate of silver painted daily over the affected parts sometimes gives great relief (one scruple of the nitrate to an ounce of water, to which ten drops of dilute nitric acid are added). Dilute nitric acid should, at the same time, be given internally, if typhoid symptoms predominate, as in the following formula:—

R. Acid. Nitrici dil., $\mathfrak{z}\text{l.}$; Syrup. Zingib., $\mathfrak{z}\text{ss.}$; Aquæ, $\mathfrak{z}\text{vss.}$; *misce.* A table-spoonful every four hours.

To arrest the spread of the process over sound skin, nitrate of silver in very strong solution, or tincture of iodine, are efficient agents. A line of circumvallation is to be painted round the erysipelatous part, so as completely to enclose it. The nitrate of silver should either be employed in the solid stick, or as proposed by Higginbottom, in solution of eight scruples of the nitrate with twelve drops of nitric acid in a fluid ounce of water. Dr. Wood has practised with success, and recommends the use of tincture of iodine.

Long and deep incisions into the inflamed textures are sometimes demanded. This is more especially the case if there be tension of fibrous tissue, such as the subcutaneous fasciæ; and erysipelas of the head is frequently greatly alleviated by repeated innumerable minute punctures, made by the point of a lancet all over the parts of the face and scalp which are affected.

THE PLAGUE.

LATIN, *Pestilentia*; FRENCH, *Peste*; GERMAN, *Pest*; ITALIAN, *Peste*.

Definition.—*A specific malignant fever which has prevailed at different times and places epidemically; attended with an eruption of a complex nature, composed of buboes or swellings of the inguinal or other lymphatic glands, and occasionally with carbuncles, pustules, spots, and petechiæ of various colours, and distributed in different parts of the body.*

Pathology and History.—Modern medicine restricts the term “plague” to a disease of dreadful severity, and of a peculiar character, which appears to have been first recognized in Egypt and in the neighbouring countries. It is impossible to determine the time when the plague first appeared in Egypt. The remotest period to which we can distinctly trace it is when spreading into other countries, as the plague of Constantinople, which broke out in 544, when Justinian was emperor. This is the first time that the disease, from its course and symptoms, can with certainty be recognized as the plague of more modern times. The symptoms were shivering and fever, at first so slight as to alarm neither the physician nor the patient; but the same day, the next day, or the day after, there appeared swellings of the parotid,

axillary, or inguinal glands, with carbuncles, and sometimes gangrene; and from the more usually diseased state of the glands it was called "*pestis inguinaria*."

The disease from that period has prevailed at short intervals in various parts of Europe as late as the seventeenth century. Sir Gilbert Blane has calculated there were no less than forty-five epidemics of plague in the seventeenth century. Fourteen of these occurred in Holland, imported, it is supposed, by the Dutch engaged in the Levant trade, about the year 1612; and twelve in England, imported, as has been supposed, from Holland. The last epidemic of plague which prevailed in both of these countries was in 1665, the year before the memorable fire of London. This epidemic was termed the "Great Plague," and spread "with such intolerable infection" that 7,165 persons are said to have died in one week, while in one year no less than 68,526 died in the city of London and its suburbs alone—an immense mortality, considering the then comparatively small amount of population.

The plague is still occasionally epidemic in Egypt, and sometimes prevails on the Barbary, Arabian, and Syrian coasts, and also at Constantinople; but it has been rarely seen out of the Turkish dominions since the seventeenth century. Nevertheless, it broke out at Copenhagen in 1712, at Marseilles in 1720, and at Moscow in 1771. In the present century it has appeared at some of the Russian Ports in the Black Sea. In 1813 it broke out at Malta and at Gozo, when the number of victims was estimated at between 4,000 and 5,000 (BURRELL). It subsequently broke out at Noja, in Calabria, in 1816; at Corfu in 1818; it appeared at Gussemborg, in Silesia, in 1819; and, lastly, in 1828-29, it devastated the ranks of the Russian army in Bulgaria; and there is reason to believe that at Odessa, towards the end of the recent Russian war, there were cases of a malignant fever, with buboes and swellings in the glands of the groin and axilla, which policy prevented calling plague.

It is believed that in this disease, as in others of this order, a specific poison, after a given period of latency, produces certain specific actions, which are either preceded, accompanied, or followed by fever. The more specific actions of the poison are the induction of a state very similar to that of typhus fever, as seen in this country; also a singular enlargement of the heart, the liver, or the spleen. But the most constant action of the poison is on the lymphatic system generally, as in typhus fever—the

cervical, inguinal, axillary, and mesenteric glands being for the most part found enlarged or otherwise inflamed, and thus giving rise to the characteristic bubo. The areolar tissue appears to be often the seat of a specific action of the poison, in the form of carbuncles: every organ and tissue of the body is likewise covered with petechiæ, and often the seat of hæmorrhagic effusion.

The extreme danger believed to attend posthumous examinations, and the prejudices of the Mohammedans, long prevented our acquiring any satisfactory data respecting the pathological phenomena of the plague; but a commission appointed by Mohammed Ali in 1834-35, and consisting of Clot Bey, Gaetani Bey, Lachesi, and subsequently of Bulard, examined the bodies of sixty-eight persons who died of the plague, and the following is a summary of their results:—

On removing the cranium the sinuses were found filled with black blood, the arachnoid veins greatly injected, and the arachnoid cavity often infiltrated with serum, and occasionally with a trifling effusion of black blood. The substance of the brain was generally less consistent than in health, and sprinkled with more bloody spots than usual. The bronchial membrane appeared sensibly inflamed, although during life the patient had presented no catarrhal symptoms. The pericardium frequently contained a reddish serosity. The serous membrane covering the heart and pericardium was often extensively affected with petechiæ. The heart, distended with blood, was almost always enlarged from a third to a half greater than its natural size, its tissues being often pale and sometimes softened.

In acute cases the stomach and small intestines were softened, and presented similar petechial appearances.

The liver was almost always larger than natural, and loaded with blood, while petechial spots were often seen at its surface. The gall-bladder was the seat of petechiæ, and in two cases blood was effused into the submucous areolar tissue.

The spleen was always twice its natural size, or even more, but was rarely the seat of hæmorrhagic effusion. It was softened, and deep in colour.

The kidneys were often found immersed in a hæmorrhagic effusion into the surrounding tissue. They were loaded with blood, and the pelvis filled with clots. The ureters occasionally contained blood, and sometimes the lumbar glands were so enlarged as to press upon them, and to account for the suppression

of urine. The bladder occasionally presented petechiæ, and occasionally the urine was mixed with blood.

Every dissection showed that buboes, wherever seated, always resulted from enlarged lymphatic ganglia, varying in size from an almond to a goose's egg. The least altered were hard and injected. In a more advanced stage some of these glands were without any change of colour, and others again as richly coloured as lees of wine, and either wholly or partially softened or putrescent. Sometimes these glands became agglomerated, forming masses which weighed two pounds or more, and around these agglomerations a hæmorrhagic effusion extended into the areolar tissue. The cervical glands often became so enlarged as to form a sort of chaplet, united with those of the axilla and of the mediastinum. The axillary glands, again, communicated with the cervical, and with those which surrounded the bronchi. Those in the groin connected themselves in the same manner with those of the abdomen, and these might be traced without interruption through the crural arch into the pelvis and along the vertebral column. It was especially among these latter that sanguineous effusion was found in the subperitoneal tissue. The mesenteric glands were often so numerous that the whole of the mesentery seemed covered with them, but they seldom exceeded an almond in size.

In the Mediterranean cities, where plague epidemics have prevailed, it is of importance pathologically to remember that epidemics of "anthrax," "carbuncle," "phlegmon," "boil," or "pustule," are not uncommon. The disease usually shows itself in the form of tumors at the lower part of the neck, between the shoulders, or in the loins. These vary in size from that of a pigeon's egg to a circumference of eight inches. They are preceded by shivering, headache, prostration, sickness, occasional vomiting, and sleeplessness, succeeded by fever, the appearance of the carbuncle, and typhoid symptoms. In some years the disease is severe, and yields a high mortality. The subjects of such disease are generally persons of sickly appearance, of gross habits of life, and who live in damp, filthy localities, where the plague formerly committed its ravages. It is extremely probable that these diseases are cognate to the true plague—that, having been born together, the source of the true plague is always in existence where these diseases prevail, requiring only the requisite unfavourable sanitary

conditions to call forth the epidemic pestilence (See *Report on the Sanitary Condition of Mediterranean Stations*, by Capt. Galton and Dr. Sutherland, pp. 88, 89).

Symptoms.—The poison of the plague produces those disordered functions of the great nervous centres which constitute the phenomena of fever, either of a low or of an active character, and sometimes so severe as to destroy the patient within one or two days, and before any secondary lesions are set up. "At Aleppo," Dr. Russel says, "in the most destructive forms of the plague the vital principle seems to be suddenly as it were extinguished, or enfeebled to a degree capable only for a short time of resisting the violence of the disease; and the form of the plague beyond all others most destructive exists without its characteristic eruptions, or other external marks considered pestilential. These cases perished sometimes within twenty-four hours."

The manner in which the disease commences varies, but generally it is preceded for a greater or less length of time by "lassitude, loss of strength, general uneasiness, and mental anxiety, to which shivering, headache, vertigo, and vomiting soon succeed; then appear the general and local phenomena, and among them the characteristic bubo, carbuncles, and petechiæ, preceded or followed by delirium or coma, too often terminating in death."

The buboes of plague seldom mature till the fever is on the decline, which rarely happens till the eighth or ninth day; nor are they generally ripe for opening till between the fifteenth and twenty-seventh day. In general, suppuration has not been so frequent as resolution, and never were the buboes seen to be gangrenous. Aubert considers the bubo as of good augury for the patient, and its suppuration as the sign of his recovery.

The carbuncle is by no means of constant occurrence, Dr. Russel having found it only in 490 cases out of 2,700. It appears more commonly in the middle or towards the decline of the disease. Hardly any external part is free from them, not even the penis; and in one instance a carbuncle formed in the throat, which was fatal. They occur more particularly on the limbs, and more especially on the legs. In some cases they form on the cheek or lips, and, by the tumefaction they cause, give to the face a hideous aspect; in others the whole of one side of the jaw has been laid bare; while in others they have formed on the eyebrow and on the eyelid, and partly destroyed the eye. Clot Bey,

however, observed they never formed on the scalp, the palms of the hands, or on the soles of the feet.

There are three different varieties of carbuncle, and all commence in the same way, or by a small red pimple, which increases, and in the centre of which a vesicle forms, containing first a yellow and afterwards a blackish serum. In the most benign the vesicle bursts, and dries up in three or four days from its first formation, the epidermis alone having been infected. The second variety involves the whole thickness of the skin, as well as portions of the cellular tissue, which is moderately tumefied, and surrounded by a dark-red areola. The gangrene in this form is circumscribed, and there results an eschar from one to two inches in diameter, which is detached by suppuration, leaving an ulcer with a sharp perpendicular edge. In the severe forms the redness and tumefaction cover a large space, and the gangrene rapidly involves the skin, the cellular tissue, and sometimes even the bones. It has been observed that the malignity of the carbuncle is in the direct ratio of the severity of the disease, but the mere existence of carbuncle is not of unfavourable augury. Their number is very various, sometimes only one, at others ten or twelve. When there are several, they often form in succession. These tumors are often very painful; and Aubert mentions one, seated on the back of an Arab soldier, four inches in diameter.

Petechiæ are observed in some seasons and not in others. They present different shades of colour, according to the intensity of the disease—rose colour, violet colour, or black. Aubert considered their appearance an almost certain sign of death. The duration of the disease is from a few hours to fifteen, twenty, thirty, or even more days.

Diagnosis.—Clot Bey says the diseases which most resemble the plague are typhus fever, severe forms of paludal fever, apoplexy, dysentery, parotitis, and scrofulous or syphilitic affections associated with febrile symptoms of a typhoid type.

Cause.—The plague, and the specific poison which it generates, seem to have a very limited geographical range. Clot Bey, indeed, considers it to be endemic along the whole of the eastern and southern coasts of the Mediterranean, the principal centres of propagation being Egypt, Syria, and Constantinople. But most authors are agreed that Egypt is the great focus of the plague, whence it may be propagated under circumstances of over-

crowding, filth, dampness, and organic decomposition. It seems determined also that the disease is often circumscribed within a very small space of country. Volney states that in Egypt the plague never commences in the interior, but always appears first on the coast at Alexandria, passes from Alexandria to Rosetta, and from Rosetta to Cairo.

All that we can safely affirm of the poison of the plague is, that it is at all times endemic in Egypt, along with the cognate diseases of "carbuncle," "anthrax," or "boil," already referred to, and every five or six years it becomes epidemic. It also appears to be, to a certain extent, influenced by season, not spreading in any very sensible degree till December, and attaining its greatest height in June, when it rapidly declines.

The period of the year, however, at which the plague prevails differs in some degree in different countries; but the total duration of the disease in any country to which it is not native appears to be inconsiderable, unless kept up by a fresh importation. At Aleppo it lasted from 1760 to 1762, a period of three years. But in Malta, Marseilles, and in the western parts of Europe, it has generally subsided in about twelve months.

In selecting its victims, this poison follows the law of most other morbid poisons, attacking the poor rather than the rich,—women rather than men,—patients labouring under disease rather than healthy individuals,—persons constitutionally feeble rather than the robust, and those addicted to intemperance, or other excesses, rather than those who more strictly observe the precepts of Mohammed. As to races—the Arab suffers more than the Negro, the Negro than the Turk, and, in Egypt, the Turk more than the European.

Modes of Propagation.—The belief that the plague is capable of being communicated is so general that it still continues to be the terror of Europe, and the ports of every nation are closed against a vessel supposed to have the plague on board. The facts by which this precaution is warranted are extremely striking; for every time the plague has appeared in Christian Europe, the arrival of a ship has been an invariable antecedent, on board of which one or more persons have died of the plague. The disease, also, invariably broke out at the port or town at which such vessel arrived; and if proper precautions were not taken, it spread into the interior of the country.

It is known that the antecedent arrival of a vessel having the plague on board, at each of the three ports of Marseilles, Messina, and Malta, and the breaking out of the disease in all those places shortly afterwards, is so remarkable that it can be only explained by admitting, in these instances, the connection of cause and effect. Moreover, the fact of the plague having originated in the preceding instances from imported cases of the disease, and not from any local influence, is demonstrated by the exemption of large bodies of persons "shut up" in the very heart of the pestilence. Thus, in the plague at Marseilles, the large nunnery of Les Dames de la Visitation Sainte Marie "shut up;" and although there was an infirmary on one side for those ill of the disease, and a burying-ground on the other for those who died of it, yet all the inmates of the nunnery escaped. The Hôpital de la Charité of the same city, a sort of poor-house, making up about 300 beds, "shut up," and escaped with complete impunity; but being converted into an infirmary for the plague patients, 200 of the poor, left in attendance, all died of the malady.

Another class of facts demonstrative of the communicable nature of the plague is the great number of persons attending on, or in communication with, the sick who die from this disease. The French army, on first taking possession of Egypt, lost no less than eighty medical officers by the plague—an immense proportion compared with the loss of the army generally. In the English army only one in forty-eight of the military died of the plague, while one-half of the medical officers died. Some few persons also have ventured voluntarily to inoculate themselves with plague-matter, and these have, with hardly an exception, fallen victims to their rash experiments.

Dr. Russel states that at Aleppo he met with twenty-eight cases of re-infection, or 1 in 157; and Clot Bey states that he and his colleagues saw many individuals perish of plague in 1834-35 who had formerly survived an attack of the disease.

Treatment.—In the treatment of the plague neither the practice of the French nor English medical officers serving in Egypt has led to any happy result; and it is to be regretted that recent experience has not in any degree advanced the successful treatment of the plague. "In the beginning of the epidemy," says Clot Bey, "when the morbid cause acts with a rapidity so great that some hours are sufficient to compromise the life of the

patient, every treatment, even the most energetic, is powerless to arrest the course of the disease. When, however, the intensity of the disease abates, we may hope for the recovery of the patient." Looking, however, to the pathology of the disease, and regarding it as a form of malignant *typhus fever*, the principles of general treatment ought to be similar to those laid down in the account of that disease.

We have no sufficient evidence to prove that plague may be carried beyond those geographical limits where it or the cognate diseases already noticed are epidemic. Quarantine establishments to prevent the transmission of such epidemic diseases are now therefore unwarrantable nuisances, and vexatious interruptions to mercantile enterprise. I am informed by a medical friend who visited Malta in 1861, that a curious instance of the wavering nature of opinion regarding the efficacy of quarantine was afforded during the last Benghazi plague. The Maltese—the most sensitive people formerly on the subject—absolutely declined to put any quarantine on arrivals from Benghazi, and trade went on as usual until the Austrians intimated that, unless Malta put Benghazi in quarantine, Trieste would put Malta in quarantine; and the poor merchants were obliged to submit. There is little risk of plague now, because of the great improvements which have taken place: and it is to be hoped that as sanitary measures are developed the barbarism of quarantine will entirely disappear; except, perhaps, where the intelligence of the people does not go beyond that of the Governor of Eupatoria, who requested that the allied armies of France and England might go into quarantine when they landed in the Crimea in 1854! (KINGLAKE.)

SECTION II.—THE CONTINUED FEVERS.

Fevers have been classified according to various theories; and much has been written on the subject. In the previous section those diseases have been described in which an eruption on the skin especially challenges attention, and with the appearance and development of which more or less fever is associated. They are described by most systematic writers as a separate class, under the name of "The Eruptive Fevers."

The "Continued Fevers," now about to be described, were at one time all recognized under the single name of "Common

Continued Fever," of which it was believed there were several varieties. But since about the year 1840 specific differences have been gradually becoming more and more obvious, so that now at least FOUR DISTINCT FEVERS can be recognized, allied by certain common characters, and not less distinctly separable by peculiar and distinctive marks. The plurality of continued fevers must now therefore be generally admitted.

The four fevers about to be described have been mixed up together in almost every epidemic, in various proportions, so that each epidemic of fever has held a peculiar character according to the nature of the dominant disease which was mainly prevalent.

The four forms of Continued Fever are now named respectively as follows:—(1.) Typhus Fever; (2.) Typhoid, Enteric, or Intestinal Fever; (3.) Relapsing Fever; (4.) Febricula.

Much has been spoken and written about the identity and non-identity of *typhus* and *typhoid* fevers; and in the first edition of this work I stated my belief that these two forms of continued fever were identical in their nature—*i.e.*, were varieties merely of a fever which resulted from one and the same specific poison. I entertained this belief partly because I had been taught as a student so to believe, and partly because I considered that the evidence then existing on the subject, and with which I was acquainted, did not fully justify any other conclusion. This belief I recanted in the second edition, for proofs of numerous and remarkable differences of a specific kind between *typhus* and *typhoid* fever have been slowly but surely accumulating since the beginning of the present century. The dissections, by Prost, of Parisian fever patients in 1804, may be said to have laid the foundations of our knowledge, and to have turned the attention of pathologists in the direction which has led to such definite results. In more than 150 dissections he always found "inflammation," with or without ulceration of the mucous membrane of the intestines. Petit, Serres, Pommer, and Bretonneau followed up the investigations of Prost; but the celebrated treatise of Louis, in 1829, was the first to give a complete and connected view of symptoms as well as of *post-mortem* lesions in the fever common in Paris; and although Prost asserted the connection of a certain intestinal lesion with a definite series of symptoms, still it was Louis alone who described this intestinal lesion

in terms sufficiently precise, and indicated with scientific exactitude the symptoms with which it is concurrent. The views of Louis were subsequently adopted by Chomel and Andral in France. In other parts of Europe, however, and especially in England, bodies of numerous fever patients were opened without finding any disease of Peyer's patches, although differences in the symptoms detailed regarding the fevers of France and England were not then so obvious. Hence arose at once two opinions, based on a *post-mortem* distinction, the "anatomical sign," described by Louis. The first opinion was that this "anatomical sign" was an incidental occurrence; or, that its occurrence was in some way connected with locality, the cases of fever being everywhere considered identical. A second opinion, however, soon began to gain ground, especially when the intestinal lesion was not found by the most careful observers in some cases where it was intentionally looked for (as in the epidemic of Toulon in 1829-30). A belief now, therefore, began to gain ground that there were in fact *two diseases* which were indifferently named *typhus* and *typhoid fever*—that one prevailed only at Paris, and the other in England, in Germany, and elsewhere, being also sometimes more or less mixed up with the Parisian fever, as measles may be with scarlet fever. Louis subsequently (1841) adopted this view.

In 1835 the "Académie de Médecine" formally proposed the question, "What are the analogies and the differences between the *typhus* and *typhoid* fevers?" The question excited considerable interest in France, but less so in England, where a strong bias has always prevailed towards a belief in the doctrine of a single fever—a belief entertained and taught by the most eminent observers and teachers of the day. But dissenters arose. Scotch, English, and American physicians, practically familiar with the fevers of their own countries, began to visit Paris to study fever there; and they were not long in learning to recognize the chief point of difference between the two fevers. Gerhard and Pen-nock, of Philadelphia, in a systematic treatise, were the first to indicate (1836) these differences, it having been already determined by Jackson and Gerhard that the fever described by Louis under the name of *typhoid fever* existed in America, and presented there the same assemblage and development of symptoms, and the same *post-mortem* lesions, as the Parisian fever.

In 1836 M. Lombard, of Geneva, after visiting London, Edin-

burgh, and Dublin, ultimately came to the conclusion that two different fevers had been confounded together; and Drs. Staberoh, of Berlin, and Kennedy, of Dublin, professed the same belief through the same medium—the *Dublin Journal*. During this year (1836), also, Dr. A. P. Stewart commenced his observations in the Glasgow Fever Hospital, where he continued his inquiry for two years. His attention was first especially directed to the study of fever by Dr. Peebles, who, during a long residence in Rome, had observed the maculæ of typhus in the contagious fever of Italy, and who first showed the difference between the characteristic eruption of fever and the cutaneous affection to which the name of “*petechiæ*” is given (*Edin. Med. and Surg. Journal*, 1835). He pointed out this eruption to Dr. Perry (then Physician of the Glasgow Fever Hospital), “and who,” Dr. Stewart states, “was the first to maintain the complete difference of the two eruptions—namely, those of *typhus* and *typhoid fever*. Dr. Stewart subsequently went to Paris and examined the fever there. The result was a complete recognition of the existence of two fevers, and of their differences—an account of which he published in the *Edinburgh Medical and Surgical Journal* for 1840, p. 289.

In 1839 Enoch Hale published an account of the fever of Massachusetts, and distinguished among them two perfectly different forms of fever, one of which agreed with the Parisian fever, while the other might be held to represent the fever described by most English writers.

Soon after this the characters of the prevalent fevers of England were noted by Shattuch (another American pupil of Louis), who published his results in the *Boston Medical Examiner*.

The appearance of these papers, and of others about this period, gave rise to an elaborate discussion of the whole question (in the pages of the *British and Foreign Medical Review*, vol. xii., p. 293); and the conclusion the reviewer arrived at seemed to favour the opinion “that the French and English fevers were varieties, that is, different developments, of a common stock, but not specifically distinct diseases.” I understand the eminent physician who wrote that review now believes in the specific distinction of the two fevers.

In America the doctrine of a specific distinction between the two fevers has been generally adopted; as represented in the

treatises of Dr. Bartlett, of Philadelphia, in 1842 and 1847, *On Typhus and Typhoid Fevers* and *On the Fevers of the United States*.

In Germany three opinions were entertained. The typhoid fever of Louis received from the Germans the name of "abdominal typhus"—thus regarding the disease as a variety of typhus fever. By some, however, it was regarded as a disease distinct from the "*typhus exanthematicus*." A third opinion also found followers—namely, that this *abdominal* typhus was the only form of continued fever—the result of a limited and narrow field of investigation.

Up till 1846 opinions were thus divided, crude, and in not a few instances quite unformed. Relapsing Fever was distinguished by some, but not by all; and all other forms of continued fever were considered in this country as identical. Under those circumstances the inquiry was taken up in 1846 by Dr. Jenner, then Professor of Pathological Anatomy in University College, and worked out by him systematically in the London Fever Hospital. There he patiently accumulated case after case of fever, until he had nearly 2,000 accurate reports before him. From these he separated all cases of relapsing fever, and then instituted a rigorous comparison of the remaining cases. He selected the fatal cases which had been examined after death, and the diagnosis of which had been confirmed. He found that he had 66 such cases and *post-mortem* examinations. Of these 66 cases, 23 had the intestinal and mesenteric lesion—the "anatomical sign" (according to Louis) of typhoid fever; and 43 cases were without this appearance. The question then remained for solution; namely—Did these 43 cases (in which the intestinal lesion was not present) differ so much in symptoms and *post-mortem* appearances from the other cases (in which the "anatomical sign" referred to was present) as to render it impossible to suppose that they were cases of the same disease? Or,—contrary to the opinion of Louis,—Were the symptoms of the two sets of cases so similar as to lead to the belief that the presence or absence of the intestinal lesion (the "anatomical sign") was a matter of little consequence?

On comparing these two groups of cases, Dr. Jenner found that while the symptoms and *post-mortem* appearances of the 23 cases were exactly the same as those described by Louis, the symptoms, course, and *post-mortem* appearances of the remaining

43 cases were entirely different—so different, indeed, as to render their separation from the other cases a matter of absolute necessity, if accuracy was to be maintained in the description of these diseases, or certainty arrived at in their treatment.

Causation, as a ground of distinction between the two fevers, is a condition upon which much stress has been laid by Dr. Jenner, and subsequently by Dr. Murchison. Dr. Jenner was the first to argue that the material media by which the two fevers are propagated are specific and different from each other, according as they are generated by the bodies of those affected with the one or the other form of fever. This argument he based upon the circumstance, that because *certain local foci* sent *typhoid* cases to the hospitals, and *certain other local foci* sent *typhus* cases there, he inferred that different specific causes existed in each focus. Dr. Murchison has also clearly stated the evidence of many other observers, which goes to prove that the two fevers have no community of origin (*Continued Fevers of Great Britain*, p. 588).

This brief history of the progress of our knowledge regarding typhus and typhoid fevers has been mainly condensed from an erudite and most interesting monograph on "The Diagnosis of Fevers," by Dr. Parkes, which appeared in the *Medico-Chirurgical Review* for July, 1851—a contribution of not less importance to science than the original investigations of those whose labours it records; for it connected the scattered observations together, and showed at once the practical value of the discovery that had been so gradually made—tending, as it did, to bring conviction to the minds of those not fully conversant with the literature of the subject, and with what had actually been achieved in different parts of the world. To Dr. Parkes, the clear, elaborate, and careful analysis he made was a labour of love—justly believing, as he does, that no subject is so important as an accuracy of diagnosis. It is the foundation of therapeutics; and he who clearly indicates how a disease can be recognized is fellow-labourer to him who points out how the disease may be cured or prevented.

This brief history teaches us how slow is the progress of discovery. The greatest discoveries have been rarely due to any single individual; but gradually, slowly, and surely the Light of Science dawns upon the world. It was so with the discovery

of the Circulation of the Blood. It was so with the discovery of the Protective Influence of Vaccination. It was so with the discovery of the Powers of Steam, and the development of the steam engine to its present condition of perfection.

Since 1851 proofs of differences between the two fevers have been still accumulating in many different directions. They especially result from the observations of Dr. William Budd, of Clifton, near Bristol; of Dr. Murchison, of the London Fever Hospital; of Professor Wunderlich, of Leipsic; and of Von. W. Greisinger, of Zurich. The observations of these two latter physicians are especially valuable, as showing the ranges of temperature in the two fevers to be distinctive of two diseases. I must here also mention the excellent lectures given by Dr. Peacock, of St. Thomas's Hospital, in 1855, and published in the *Medical Times* of 1856, "On the Varieties of Continued Fevers and their Discrimination," as influential in forwarding these modern views, for they appeared at a time when the specific distinctions between typhus and typhoid fevers were less generally admitted than at present. Thus the evidence has slowly but surely accumulated; and when the whole subject has been re-examined in all its relations, the conclusion irresistibly forces itself on the understanding, that a belief in the identity of *typhus* and *typhoid* fevers is no longer tenable.

In common with many, I had, as a student, been taught to recognize the striking similarity between the two fevers, in outward aspect, in many respects; and therefore I was unduly biassed by the *resemblances*, rather than led to give sufficient importance to the numerous and remarkable *differences* between them which are now to be described.

With regard to their most prominent points of resemblance and difference, it may be shortly stated here, that all the points in which the two fevers agree are common to them and many other diseases, and therefore are of no value as *indicia* of a species. On the other hand, the points in which they differ are all of a very special nature. The points in which they agree may all be summed up in the phrase "typhoid symptoms"—a set of symptoms which are met with in a great variety of diseases, and therefore are of no specific value in the question at issue. These so-called typhoid symptoms (represented by the phenomena of stupor, low delirium, general prostration, *subsultus tendinum*, a

dry and encrusted mouth, deafness) occur not only during the course of *typhus* and *typhoid* fevers, but are also found to occur and to group themselves in a similar manner in *pycæmia*, *uræmia*, some forms of *pneumonia*, and in many cases of *acute tubercle* (W. BUDD).

ENTERIC FEVER—Syn., TYPHOID FEVER.

LATIN, *Febris enterica*; FRENCH, *Fièvre typhoïde*; GERMAN, *Abdominal-typhus*—Syn., *Ileo-typhus*; ITALIAN, *Tifo enterico*.

Ileo-tifo
Definition.—A continued fever associated with an eruption on the skin of rose-coloured spots, chiefly on the abdomen, appearing generally from the eighth to the twelfth day, occurring in crops, each spot continuing visible about three days. Languor and feebleness are prominent from the first, attended by headache, abdominal pains, and (early) by spontaneous diarrhœa. With the advance of the disease the diarrhœa increases, the discharges being for the most part liquid, copious, of a bright yellow colour, devoid of mucus, occasionally containing altered blood; in re-action alkaline, and containing a large proportion of soluble salts and some albumen. The disease may terminate favourably by a gradual restoration to health during the fourth week. The average duration of the fever is about twenty-three days. Death in the majority of fatal cases occurs towards the end of the third week. There are symptoms also associated with the characteristic lesion of this form of fever—namely, fulness, resonance, and tenderness of the abdomen; more or less tympanitis, with entire effacement of the natural lineaments of the belly; gurgling in the iliac fossæ; increased splenic dulness. The specific lesions are enlargement of the mesenteric glands, with deposit in the glands of Peyer and in the minute solitary glands of the small intestine.

Pathology and Symptoms.—Typhoid fever begins gradually—often, indeed, so very insidiously that its commencement is not always able to be fixed.

This form of continued fever is described under a great variety of names, by various writers, such as *typhus mitior*; *nervous fever*; *abdominal typhus*; *common continued fever*; *entero-mesenteric fever*; *dothi-enteritis*; *follicular enteritis*; *bilious fever*.

The fever may be ushered in with rigors, chilliness, or profuse diarrhœa; and amongst the early symptoms, the most characteristic are the abdominal pains and diarrhœa, which continue to

low fever

increase. The countenance indicates anxiety, the mind continues clear; but delirium, when present, is generally active. The patients are vivacious, and disposed to leave their beds. The conjunctivæ are pale, the pupils dilated, the cheeks somewhat flushed, and epistaxis not seldom occurs, at repeated intervals, during the first week. The belly enlarges, as in mesenteric disease, and is resonant on percussion. Gurgling on firm pressure may commonly be detected in the right iliac fossa, and there is often tenderness in the same situation. From the seventh to the fourteenth day the characteristic eruption appears. As a rule, the flushing of the face is more marked towards evening; but the complexion does not get muddy, as in typhus, and the flush of the cheeks is bright and pinkish—not dark red—and is often circumscribed, and then strongly contrasts with the surrounding pale skin. During the third week the abdomen becomes more distended; the diarrhœa increases, the stools often amounting to *five, six, or even eight and ten* a day. They are liquid, pale brownish-yellow, with flocculi of an opaque whitish-yellow colour floating through them like coarse bran, and as the patient loses strength they are passed involuntarily. Pain is rarely complained of unless perforation of the gut occurs; and hæmorrhage from the bowel is an occasional symptom during the third or fourth week. The frequency of the pulse often varies much from day to day, without any appreciable coincident alteration in the general or local symptoms. It is generally soft. The tongue at first is red and fissured, but ultimately becomes dry and covered with a pale-brown fur. The splenic dulness is generally increased. Pulmonic complication is not uncommon. *generally tracheitis.*

X In cases that recover, a remarkable fatuity remains behind long after recovery; and there appears to be some diminution of intellectual power for some time after convalescence is restored. Dr. Jenner has seen many cases in which childishness of mind remained for more than a month after apparent restoration to health. The patient generally wakes up, as it were, from the fever a complete imbecile. The whole man is changed. He seems to have renewed his youth. Childhood and infancy return, and the greatest care is necessary to prevent untoward events. NO MAN CAN BE CONSIDERED AS FIT FOR WORK, OR FOR GENERAL MILITARY SERVICE, FOR THREE OR FOUR MONTHS AFTER AN ATTACK OF SEVERE TYPHOID FEVER.

With regard to the symptoms generally of typhoid fever, it is of great practical importance to be constantly alive to the fact that no necessary connection exists between the intensity of the general symptoms of the disease and the extent of the intestinal mischief which may supervene, or the absolute danger of the case. Two cases, out of several related by Dr. Bristowe, show that the patients (men) carried on their daily avocations, so mild *seemed* the disease to be, up to the very moment of fatal perforation of the gut. Indeed, the most suddenly fatal cases seem to be the very cases in which strongly marked febrile phenomena do not occur. In a case related by Dr. Murchison, a man twenty-one years of age died on the twenty-fifth day of the fever. Up till the twenty-third day there were no symptoms to indicate danger. He suffered from very slight diarrhoea; the pulse seldom rose above 90; and the patient could get out of and into bed. About forty-two hours before death the pulse rose to 120, associated with sudden pain in the lower and right side of the abdomen. Profound collapse indicated that perforation had ensued, and death soon followed. The *very slightness of the symptoms* ought, therefore, to rouse suspicion, knowing, as we now do, that, associated with the characteristic eruption, the following *four sets of phenomena* may be all that precede a fatal hæmorrhage or peritonitis; namely,—(1.) An elevation of temperature towards evening of only 1° or 2° above 98° Fahr.; (2.) Moderate increase towards evening in the fulness and quickness of the pulse; (3.) A little headache during the first six days; (4.) Scanty urine.

Again, the physician must keep in view the fact that relapses of all the symptoms, including the eruption, not unfrequently supervene. He must not be betrayed into the belief that danger is past, if, towards the eighth or tenth day, the little headache that prevailed may pass away, and the other febrile phenomena just mentioned may subside. It is on record that events such as these have led to the belief that convalescence from a mere “febricula” had been established, leading to the discharge of the unfortunate patient from hospital. His vocation, if a soldier, would then compel him to undertake severe duties during the actual height of a severe disease, made more dangerous and perhaps fatal by such a mistake.

Another symptom, often very painful, is *meteorism*, or the accumulation of air in the large intestine. This is present

in a greater or less degree in one-half of the cases, and when considerable it always marks a grave affection, and one generally fatal. On the contrary, the abdominal muscles are, in a few cases, tense and strongly contracted. It is, however, the experience of all physicians that there is no condition so low, and no symptoms so severe, from which the patient may not recover; and, on the other hand, there is no case of this form of fever so slight that it is to be considered free from danger. The prognosis must therefore be cautious, because perforation of the intestine may follow the mildest case, and death from peritonitis ensue.

The symptoms of typhoid fever cannot be said to be fully expressed till the characteristic eruption has appeared.

*8th day
fever*

The Eruption consists of the so-called *rose spots* peculiar to typhoid fever, the "*taches rosées lenticulaires*" of Louis. They begin to appear from the sixth or seventh to the twelfth or fourteenth day of the disease, very rarely later, and still more rarely at an earlier period than the sixth day. A very delicate scarlet tint of the whole skin, closely resembling the skin of a person soon after leaving a hot bath, sometimes precedes, by a day or two, the characteristic eruption of typhoid fever; and this is important to remember, because it may be mistaken for the rash of scarlet fever, especially if sore throat is present. The eruption consists of slightly elevated papulæ or pimples; but, to detect their elevation, the finger must be passed very delicately over the surface of the skin, because, although pimples, they are not hard, like the first day's eruption of small-pox. Their apices are neither acuminate nor flat, but invariably lens-shaped or rounded, and the bases gradually pass into the level of the surrounding cuticle. No trace of vesication can be detected on their apices. They are circular, and of a bright-rose colour, the colour fading insensibly into the natural hue of the skin around. Their margin is never well defined. *They disappear completely on pressure,* resuming their characteristic appearances as soon as the pressure is removed. These characters they preserve from their first appearance to their last trace. They leave behind no pit, scar, or stain. They vary in size, but their usual diameter is nearly 2 lines, but varying from 1 to $2\frac{1}{2}$. The ordinary duration of each papula is about two days, but its existence varies from two to six days, and fresh ones generally make their appearance every day or two after the first appearance of eruption, and they continue to appear in successive crops till the

twenty-first or twenty-eighth day of the disease. Sometimes only one or two are present at first, after which one or more fresh ones make their appearance. The eruption of such spots does not consist of a great number at one time—only from six to twenty. The eruption occupies usually the abdomen, thorax, and back; but is sometimes present on the extremities, and is sometimes, though rarely, so thickly seated that scarcely an interval of normal cuticle is left between. THIS SUCCESSIVE DAILY ERUPTION OF A FEW SMALL, VERY SLIGHTLY ELEVATED, ROSE-COLOURED SPOTS, DISAPPEARING ON PRESSURE, EACH SPOT CONTINUING VISIBLE FOR THREE OR FOUR DAYS ONLY, IS PECULIAR TO AND ABSOLUTELY DIAGNOSTIC OF TYPHOID FEVER.

The eruption is, however, often so scanty that the physician may justly hesitate for a day or two to make a diagnosis. The first crop of the eruption is rarely quite decisive; but as soon as successive crops, even of two or three spots each, appear, all doubt is removed. When the eruption is scanty, it is advisable to surround each individual spot with an ink line, in such a way as to distinguish accurately the period of its appearance (W. T. GAIRDNER). It is the occurrence of this eruption which clenches the diagnosis; and which becomes absolute, as regards typhoid fever, when, in a febrile disease attended by diarrhoea, or simply looseness, unequivocal rose spots appear on the sixth or eighth day. If they do not appear, then the diagnosis cannot be said to be complete till the case has been watched for several days, and the age of the patient and the history of the illness has been fully and carefully studied. In children between *one* and *five* years of age the phenomena do not seem to be so easily observed as in adults.

It has been now clearly established that typhoid fever is by no means an unfrequent disease amongst children, and has been often described under the name of "*Infantile remittent fever.*" Boys seem to be more liable to attack than girls. It is most frequent between six and eleven years of age; and from five to nine seems the period of greatest liability. Its occurrence is rare during the first years of life. Nevertheless, it is on record at the following very early ages:—namely, between two and three months; three months; six months; seven, ten, and thirteen months (WUNDERLICH, HENNIG, FRIEDRICH, RILLIET). The author of a very interesting Review on the typhoid fever of children, in the *British and Foreign Medico-Chirurgical Review*

Fastigium of Temp 3rd & 4th day in typhoid
before or on 9th day in typhus

for July 1858, p. 161, mentions, in his own experience, the occurrence of typhoid fever in a girl one year and seven months old; and also in a boy two years of age.

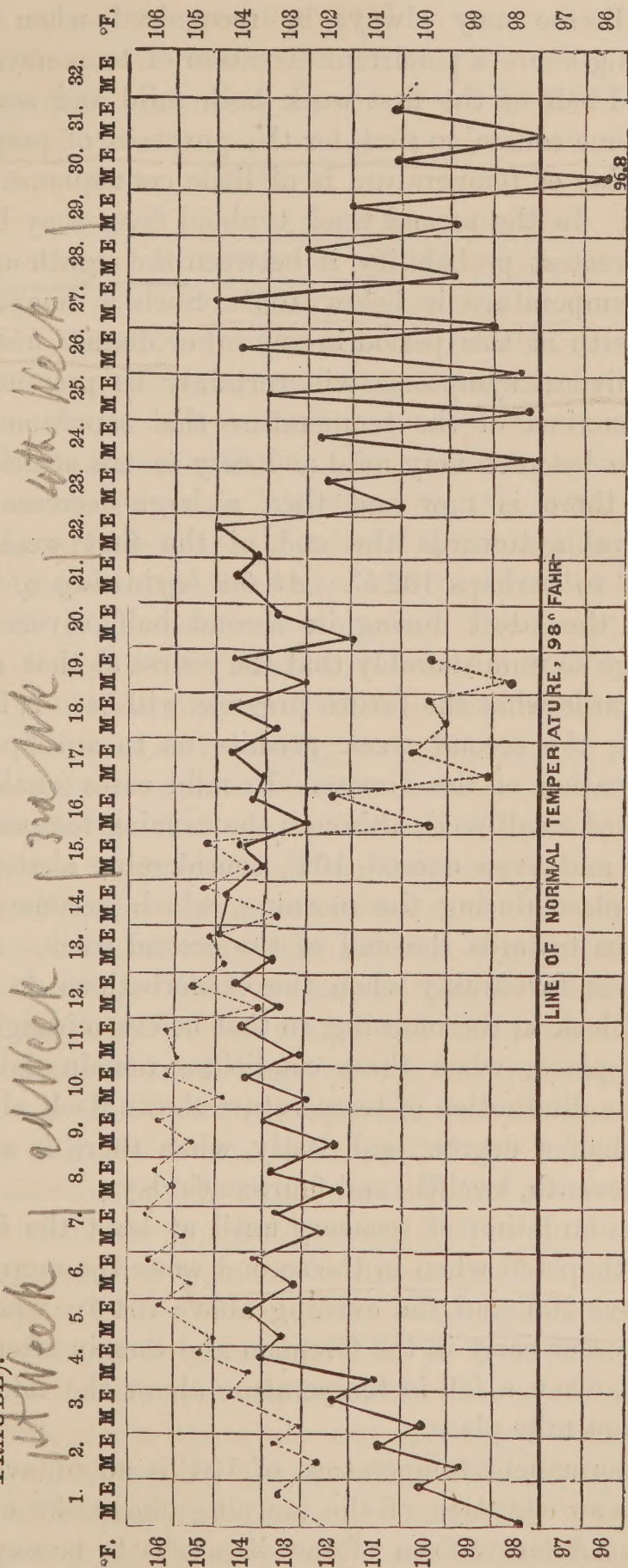
Typhoid in children
The chief symptoms of typhoid fever in the child are,—splenic enlargement, diarrhoea, meteorism, gurgling in the course of the colon; associated with pyrexia, quickened respiration, bronchial catarrh; delirium, somnolency. The eruption already described, and sudamina, are nearly constant in children after five years of age. The rose-coloured spots are especially frequent on the back and the extremities, so that, if the abdomen and chest only are examined, their presence may often not be apparent.

The Temperature during Typhoid or Intestinal Fever.—Wunderlich has given a summary of results derived from the observation of 700 cases of typhoid fever, investigated thermometrically (*Arch. der Heilk.*, vol. ii., 1861, p. 433; also, *Edin. Med. Journal*, Nov., 1862, p. 465).

The course of the disease is typical, and the type is characteristic; and when irregular cases occur, irregularity may in general be traced to a special cause. The mode of accession is pretty nearly the same as in much more severe cases. Increase of temperature in the evening and remissions in the morning follow one another for about three days, the temperature every morning and every evening being about 2.2° Fahr. higher than on the preceding morning and evening, while the morning temperature is generally about 1.1° lower than that of the previous evening; or, according to the following formula:—

First day, morning, 98.5° ; evening, 100.5° : *second day*, morning, 99.5° ; evening, 101.5° : *third day*, morning, 100.5° ; evening, 102.5° : *fourth day*, morning, 101.5° ; evening, 104° . In the second half of the week the evening temperature is from 103° to 104° ,—the morning temperature about a degree lower. On the third or fourth day the fastigium or height of the fever is attained, when the temperature in the evening amounts at least to 103.5 Fahr. From that time onwards the fever proceeds in regular stages of weekly and half-weekly periods. When the temperature on the first or second day reaches to 104° , or where in a child or in an adult the evening temperature between the fourth and sixth days does not rise to 103° , where, in the second half of the first week there is considerable abatement of the evening temperature, we have in such cases certainly not to do with typhoid fever; on the other hand,

TYPICAL RANGES OF TEMPERATURE IN CASES OF TYPHUS AND TYPHOID FEVER CONTRASTED WITH EACH OTHER THROUGHOUT THEIR COURSE, FROM THE BEGINNING TO THE END OF THE DISEASE. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS. The dotted lines indicate the *Typhus* range; the continuous dark lines indicate the range in *Typhoid* (WUNDERLICH and TRAUBE).



*Mild & severe cases diverge unmistakably
at beginning of second week*

X the disease may always be recognized when there is in the evening hours a persistent elevation of temperature. During the second half of the first week both mild and severe cases follow the same course, so that, for the purposes of prognosis, the determination of temperature is of little consequence during the first week. In the second week typhoid fever may be excluded with the greatest probability, if between the eighth and eleventh days the temperature is below 103° . Such a temperature is rarely met with at this period in any other disease, and where it occurs unequivocal symptoms will certainly be present. It is only in the maxima of the temperature that sometimes a difference is visible between very mild and very severe attacks. In the mild cases there is now and then a large decrease of temperature observable towards the end of the first week—namely, from 105.8° to perhaps 102.5° . *MB* At the beginning of the second week, or at the latest during its second half, severe and mild cases diverge so unmistakably that the course at that period is decisive as regards what the future progress will be. A favourable course during the second week permits us to anticipate a favourable termination of the disease. In mild cases (analogous to those of modified small-pox), although the evening temperature may reach 103° ; and even exceed 104° , considerable abatements (1° to 2°) take place during the morning, which become more and more obvious towards the end of the second week. Such mild cases progress favourably when the exacerbations do not begin before ten o'clock in the morning, so that before midnight an abatement takes place; when these conditions remain daily the same, or when a diminution of temperature shows itself, although not more than half a degree; and, lastly, when there is an abatement on the eleventh, twelfth, and fourteenth days.

A retardation of recovery until at least the fourth week is to be anticipated when in the second week the morning temperature is above 103° and the evening above 104.5° ; when the exacerbations occur early in the forenoon and remain after midnight; and, lastly, when a fall in temperature about the middle of the week does not take place.

X A permanent temperature of 104° is an unfavourable sign—so also is an elevation of the morning above the evening temperatures. A severe form of the disease is to be expected when the morning temperature at the beginning of the second week is

above 104° , and when the evening reaches nearly 106° ; and when towards the end of the week a rise still takes place. The most unfavourable cases are those where, in addition to these unfavourable conditions, oscillations are added, even if these consist in diminution of temperature.

In the third week the patient enters upon those highly characteristic quotidian vacillations of 4° , 6° , and even more degrees Fahr. between the morning and the evening temperatures. If the case is mild, the evening exacerbations gradually decrease in intensity, and the morning temperature is regularly at first from 3° to 4° below the evening. The fever ceases in the course of the week, the temperature reaching its natural standard, and convalescence commences, as a rule, sometimes in the third week, generally in the fourth week, or at the latest in the fifth week.

In severe cases the characteristics mentioned as peculiar to the third week already commence in the second. The temperature in the mornings is high (104° Fahr., and more), and differs but little from that in the evening; or even that high temperature increases in the afternoon and evening to a still higher degree. In this it differs from a remission of the fever in a mild case, inasmuch as in remissions the heat in the mornings sinks below the average degree of temperature in typhoid cases—*i. e.*, below 103.3° Fahr. to 104° Fahr. In severe cases, on the contrary, the temperature always remains above the average degree, and rises still higher in the evening. Real remissions in such cases are not met with during the whole of the second and third weeks; but when the case is favourable, although severe, the temperature is about a degree lower than in the second week, and the remissions do not take place till the fourth week; and if the temperature remains as high, or rises higher than it was in the second week, the remissions do not occur till the fifth week, and irregularities in the ranges of temperature always render the prognosis doubtful.

So late as *the fourth week* the evening temperatures are still high, and they decrease very gradually even in favourable cases. Towards the end of the fourth week, or in the fifth week, or even so late as *the sixth week*, the great and increasing remissions commence—a period at which various other phenomena occur, and when the complications and dangers are numerous.

The complications generally make their appearance about the third week, and threaten or tend to a fatal end up to the very

beginning of convalescence. In the mild types the growths in the intestinal glands are no doubt such as are eliminated by mere rupture of the vesicles, which simply heal without ulceration. The severe cases owe their severity partly to the more extensive growth of new material in the vesicles of Peyer's glands, partly to the mode of elimination of that material; the healing of the parts being accomplished under great excitement of vascular re-action, renewed hyperæmia, sloughing, softening, and final cicatrization.

Cases intermediate in severity between the mild and severe cases just described are not unfrequently met with. Many of them, although they show a course more or less irregular, nevertheless follow a pretty clearly defined type as to variations of temperature, and are capable of clinical recognition. There are still considerable evening exacerbations during the second week, yet with a tendency to abatements in the mornings. During the third week great vacillations between morning and evening temperature continue, and sometimes also between single days. During the fourth or fifth week the normal temperature is reached in the morning; but it is only in the fifth or sixth week that the temperature becomes permanently normal—the evening temperature showing a complete freedom from fever—so that the beginning of convalescence can only be established with certainty by the use of the thermometer.

In the majority of cases of typhoid fever, severe as well as mild, a peculiar periodicity of weeks and half weeks cannot be mistaken. Each week shows a distinct character, which cannot be overlooked in a graphic representation. On the first and last days of each week changes generally take place which are either temporary changes or continue till the fever subsides.

Duration of Attack and the Mode of Recovery, or the transition into the feverless state, is peculiar and characteristic of enteric fever. With rare exceptions, the defervescence is a remittent one. The great vacillations between morning and evening recur for a longer or shorter interval. For weeks the evening temperature may amount to 104° Fahr. or more, whilst in the morning the patient is quite free from fever. At the same time the transition into the feverless condition may follow different courses. The remissions may either become longer and longer—the morning temperature decreasing and the evening remaining stationary; or

after some time the remission may become shorter and shorter—the evening temperature, together with the morning temperature, gradually descending. Again, the differences between the morning and the evening temperatures may remain nearly the same, while a relative decrease takes place at both periods; or the fever shows a sudden transition into the remissions with low temperatures—changes which generally correspond with the commencement of weeks. The period of development of the disease occupies two weeks, or a week and a half in slight cases; in severe cases it may occupy two and a half to three weeks. The initial stage (that is, the period when the growth of material in Peyer's patches takes place) lasts about half a week. The removal or elimination of the growth may take place in a week; but the process may extend over several weeks. In mild cases the disease continues at its height for only a week or a week and a half,—rarely for two weeks; so that the whole duration of a mild case of typhoid fever extends from eleven to eighteen days. The period of convalescence occupies from one to two weeks. The whole disease, therefore, in mild cases, may be gone through in from three to four weeks,—rarely in two weeks and a half.

In severe cases the disease continues at its height for from two weeks and a half to three weeks and a half. Then an undecided period of irregular duration succeeds, after which decided abatement is established, the defervescence occupying a week, followed by another week of convalescence. Consequently, the whole disease extends from four and a half to ten weeks, or even longer. Regarding the mean duration of illness in *typhoid fever* considerable differences of statement are to be found—a circumstance not to be wondered at when the nature and seat of the pathognomonic lesions of this form of fever are recognized as influencing the duration of the disease.

Dr. Shattuck assigns the mean duration of <i>typhoid fever</i> to be	22 to 24 days.
The mean duration of the Parisian cases of 1839-40 were	19.6 „
Dr. Jackson's experience in America gives	22 „
Dr. Jenner's experience in London leads him to give	21 to 30 „
Dr. Murchison	24.6 „

The mean of these varied statements gives nearly twenty-three days.

Immerelapses { Most common in autumn
more severe than 1st attack
occur about 10-14 days after

It is now well known that during the progress of this form of fever there is a repetition of the development of new material in the individual gland-vesicles of the intestine, and consequently a succession of retrograde metamorphoses; so that, in many extreme cases of *typhoid* fever, it is not unusual to have the malady prolonged throughout a course nearly double as long as that of *typhus*; and that, undoubtedly, the influence of the secondary local lesions of *typhoid* fever is great in protracting the disease. Thus it is that a very indefinite idea of its duration prevails; and, as Dr. Jenner has shown, it is of the greatest importance to know when the original fever ceases, after which we are to consider the subsequent symptoms as due to the effects produced by the local lesions.

As long as fresh eruption continues to appear, the fever cannot be regarded as having terminated; and, except in cases of relapse, fresh spots never appear after the thirtieth day (JENNER) or thirty-fifth day (MURCHISON).

Case Jenner Sept 70 True relapses are occasionally observed. They occur about ten days or a fortnight after convalescence from the first attack, and are marked by a return of all the former symptoms; while the duration of the attack is usually shorter than that of the first; and, according to the experience of Murchison, it is more severe. Such relapses are most common in autumn.

In particular cases following a spontaneous course, and still more in cases treated with calomel, a considerable shortening of the whole febrile period will not only be observed, but some peculiar modes of defervescence will occur. The temperature is reduced where calomel acts beneficially; and the beneficial remission is persistent.

The influence of hæmorrhage from the bowels in reducing temperature has been also well shown in a case recorded by Dr. Parkes. It occurred in a female twenty-five years of age. Diarrhœa was considerable; and blood was largely passed in fluid stools the night before the seventeenth day of the fever. On the morning of the seventeenth day the temperature was as low as 93° Fahr., rising in the evening to about 101° Fahr. After the eighteenth day diarrhœa ceased; but the differences between the morning and evening temperatures continued to be very great; and it was not till the twenty-sixth day that these differences began to grow less and less.

Water & Urea diminished
Urea & uric acid increased
H₂SO₄. H₂PO₄ unaltered

The approach of death is indicated by a permanent or persistent elevation of temperature in the morning (as high as 106°); by a sudden rise to 108°, or even higher; and more seldom to a depression below 93°.

Condition of the Urine in Typhoid Fever.—It is not till the third or fourth day of the fever that the urine assumes any special characters. It is peculiar in the following respects:—

I. As to normal constituents:—(1.) *The water* is greatly diminished, generally about one-half or even to one-fourth or one-sixth. This lessening of the water is most marked during the first week; it then begins to increase gradually during the second and third weeks; and at the end of the fourth week, in favourable cases, it has reached its normal standard. (2.) *The whole amount* of the urine does not seem to stand in any close relation to the febrile heat; but when the temperature begins to fall permanently, the urine increases at once, or very soon after. (3.) *The specific gravity* of the urine is high in almost all cases where the urine is scanty; and at convalescence the specific gravity diminishes, sometimes before the amount of water increases; *i. e.*, at convalescence the lessening of the solids of the urine is often prior to the increase in the water. (4.) *The urea*, as a rule, seems to be augmented, during the febrile period, above the physiological standard proper to the individual; and it sinks again below this standard during convalescence. The amount of increase varies: Vogel has noted 78 grammes, or 1,200 grains, in 24 hours; while Parkes has noted 57 grammes, or 880 grains, in that time. In most of the cases observed by Dr. Parkes the average increase has been about one-fifth above the physiological standard proper to the individual; and the augmentation is most marked in the first week, when the water and the chloride of sodium are at the lowest point; and if the fever be continued beyond the third or fourth week, the urea keeps up in amount. The relation of urea to temperature is yet uncertain. (5.) *The chloride of sodium* is diminished (indefinitely); the cause of the diminution being in part due to the lessened ingress of this substance on account of spare diet: or due to the elimination of large quantities of it with the stools or the sweat. (6.) *The uric acid* is uniformly increased in amount; and it is relatively greater than that of the urea. It is often doubled in amount; and the increase progresses up to the fourteenth day, when it is at its greatest. It then diminishes to

the twenty-first or twenty-eighth day; and during convalescence falls below the normal amount. Spontaneous deposits of urates occur very frequently; and when there is no such deposit it may be brought about by a drop of acid; but as yet the fact has no particular significance. (7.) *The sulphuric acid and phosphoric acids maintain their amounts very much the same as in health; and sometimes a little above that: and seeing that much of the former is derived from food, its abundance in typhoid fever would indicate active tissue-change, when little or no food is being taken.* (8.) *The pigment at first is sometimes enormously increased, measured after Vogel's method (by comparison with a scale of colours). It has sometimes amounted to 80 or 100 in 24 hours, the normal amount being 3 to 6 (VOGEL). This, Dr. Parkes says, is to be referred to increased disintegration of blood-cells: it is therefore much more highly coloured than the mere concentration will account for.* (9.) *The acidity of the urine appears always great during the early period, simply from concentration; but by neutralization with an alkali it is found actually to be below the average by one-fifth, or even by one-fourth. During the third week the acidity still continues to lessen; and ultimately the urine may even become alkaline from fixed alkali. It may also become alkaline from ureal decomposition, soon after being passed. Therefore it is necessary, in all observations on this point, to distinguish carefully between the alkalinity due to fixed alkali and that due to ammonia.*

II. As to abnormal constituents:—(1.) Albumen occurred in 33·3 per cent. of the cases examined by Dr. Parkes. In 23 per cent. of these cases it was temporary, and entirely disappeared before the patients left the hospital. In the other cases it was permanent; and in one of these a very profound kidney lesion which had not previously existed was immediately excited by the fever. (2.) *Renal epithelium, casts, and blood are sometimes seen in the cases with temporary albuminuria—the blood generally in microscopic quantities; although in bad cases it may be greater in amount. Different phenomena in the course of typhoid fever variously affect the urine. The effect of diarrhoea is to diminish both water and solids, the chloride of sodium especially. Non-excretion of urea, or deficiency in its solid matters, often coincides with the putrid, adynamic, or profound "typhoid" state, and with symptoms which imply more or less blood poisoning from reten-*

tion. Local lesion in the kidney may lead to this, or, from failing circulation, less blood may pass through the renal vessels, or there may be, as Dr. Parkes suggests, some special condition or combination of urea which hinders transudation. Such non-excretion is most apt to supervene during the third or fourth week, when the first stage of the disease is over, and when the growths in Peyer's glands, and in the mesenteric glands, are softening, when the secondary blood poisoning occurs, and when the heart's action tends most to fail.

Judging from the urine alone, the febrile action appears strongest in the first week of enteric or intestinal fever (typhoid), although the temperature is highest in the second and commencement of the third week.

The prognosis in severe typhoid fever appears to be more favourable in proportion to the free excretion of urea and uric acid (PARKES). The excretion of these effete products is a most necessary point; for there is more danger *in the retention* than in any amount of fever and *formation of them with elimination*. The greater the excretion in typhoid fever the better; and as long as 500 to 700 grains of urea in men, or 300 to 500 in women, are being passed in each twenty-four hours, the progress so far is favourable. But whenever, while the fever continues, the urea falls much below these amounts, we may anticipate a low typhoid condition, or some local inflammation, as pleurisy, which may relieve the blood for a time from some of the effete products, but which at the same time may kill the patient.

The existence of slight albuminuria or hæmaturia is not of itself unfavourable; but if either be in large amount, or if there be exfoliation of epithelium or renal cylinders present in the urine, retention of urea and its consequences may be expected.

Morbid Anatomy of the Lesions in Typhoid Fever, with special reference to the Phenomena and Progress of the Disease.—The abdominal complications of typhoid fever, as they are sometimes called, are mainly due to lesions of the solitary and aggregate glands of Peyer, and to enlargement of the mesenteric lymphatic glands. This lesion in the ileum is especially recognized as the "anatomical sign" of enteric or typhoid fever. It is necessary to remember, however, in connection with the age of typhoid fever patients, that the solitary vesicles and the aggregate glands of Peyer are known to be most fully developed and most active

in youth, up to the age of early manhood; after that time they begin to disappear, and are obviously less active in the adult after thirty years of age. Structure and function seem to be alike impaired by age, till at length, after forty or forty-five years, traces only of their existence are apparent, or they have altogether disappeared. The gland substance (whose structure has been so well described by Dr. Allen Thomson, Kölliker, and Boehm) no longer exists; and the places where the patches of Peyer once were may be detected only after careful examination, —a mark of varied form and character being all that indicates the place of the patch. There is therefore a good anatomical reason why typhoid lesions are rarely found after fifty years of age, and seldom after forty. Dr. Jenner records only three cases beyond fifty—namely, one at fifty-one and two at fifty-five. Dr. Wood has observed one case at fifty-five years of age. Dr. Murchison notes two cases above sixty-five, and refers to five other cases between sixty and seventy-five, related by MM. Lombard and Gendron. Dr. Wilks refers to the case of a woman aged seventy, of very doubtful history (*Path. Society*, 1861). These exceptional cases are explicable when it is known that the existence and functional activity of these glands are sometimes prolonged for an indefinite term of years beyond the usual period of their existence. On the other hand, it is in childhood and early life that these glands are most obvious, and their functional activity the greatest; and therefore it is extremely significant to find that “more than one-half of the cases of typhoid fever occur between fifteen and twenty-five years of age; and in very early life the proportion of cases of typhoid would be greater were it not that many children labouring under this disease are described as cases of ‘Infantile Remittent Fever’” (MURCHISON).

The following records with regard to the age of typhoid fever patients, collected by Dr. Murchison, demonstrate these points:—

PER CENTAGE OF CASES OF TYPHOID FEVER AT DIFFERENT AGES.

Under Ten Years,	.	.	.	.	.	.	.	6·04
„ Fifteen Years,	.	.	.	.	.	.	.	20·14
From Fifteen to Twenty-five Years,	.	.	.	.	.	.	.	52·08
Twenty-five Years and upwards,	.	.	.	.	.	.	.	27·76
Thirty,	„	„	.	.	.	.	.	14·22
Forty,	„	„	.	.	.	.	.	5·19
Fifty,	„	„	.	.	.	.	.	1·46
Sixty,	„	„	.	.	.	.	.	0·5

Age 21 $\frac{1}{4}$ yrs

The average age of typhoid fever cases is $21\frac{1}{4}$; and the fever is pre-eminently a disease of childhood and adolescence.

Lesions in Typhoid Fever.—Of these the most noticeable are to be seen in the intestines, and may be considered in the following stages:—

I. *A generally congested state of the mucous membrane of the intestines, especially expressed in the vicinity of the solitary glands, which are surrounded by vascular rings, and clustered groups of vesicular glands which constitute Peyer's patches.** This vascularity seems to be very general, involving more or less of the abdominal viscera. Sensations of heat and abdominal distress are associated with this morbid state, and the lineaments of the belly are obliterated.

II. *Associated with this congestion, the gland-vesicles become obviously prominent.* Increased growth of the gland-cells occurs, till the closed sacs of the glands become filled up with crude

* The following tabular statement of the anatomical forms of the glands which compose the substance of the mucous membrane of the alimentary canal is mainly condensed from a careful description of them by Dr. Allen Thomson, Professor of Anatomy in Glasgow, published in Goodsir's *Annals of Anatomy*, vol. i., p. 33, and from the descriptions of Kölliker. The nomenclature is definite, and distinctive of the various forms of the glands; and it will be adhered to in all the future descriptions of lesions of the mucous membrane of the intestines. Much confusion prevails from the indifferent use of the terms vesicle, tubule, follicle, &c., as applied to the mucous glands in descriptions of the lesions in dysentery and fevers; therefore it is considered necessary to explain exactly, at the outset, the nomenclature adopted in the text.

I. VESICULAR, LENTICULAR, or PIMPLE-LIKE GLANDS—Usually closed.

(a.) Solitary—*e. g.*, in the palate, buccal membrane, œsophagus, and stomach; also found deeply imbedded in the great gut, and scattered more near the surface of the small gut.

We know nothing about the comparative abundance of these glands in a healthy intestine; nor are we certain whether or not they disappear after a certain age, like the vesicles which compose Peyer's patches. Many of the so-called solitary glands seen in disease may be in reality new formations. At all events, they occur in much greater numbers in certain diseases than their known frequency of appearance in the healthy intestine would lead us to expect.

(b.) Clustered in groups—*e. g.*, Peyer's patches of glands in the ileum.

II. FOLLICULAR OPEN GLANDS or CRYPTS—A transient condition of the vesicular glands, after rupture and discharge of their contents—*e. g.*, great gut and stomach.

III. TUBULAR GLANDS—Occur in the small and large intestines, as the so-called follicles of Lieberkühn; and in the stomach, as the stomach tubes.

IV. RACEMOSE GLANDS—Consisting of tubes with simple sacs or vesicles, in clusters round a stalk or duct—*e. g.*, the cardiac œsophageal glands, and the duodenal glands of Brunner.

material. This condition is sometimes described as *intumescence* of the glands, and with the congestion just noticed constitutes the stage of "*infarction*," as described by the older authors. Symptoms denoting intense irritation of the mucous membrane—*catarrhal* and *gastric* symptoms—prevail; and the mucous membrane generally is swollen and turgid, especially the villi of the intestines, which are particularly distinct, imbedded in a thick layer of dirty-yellow gelatinous mucus. Although these conditions seem to involve the whole of the mucous membrane in the first instance, yet they soon begin to be more expressed towards the lower end of the small gut than in any other part. The time at which this increase of cell-growth commences in these glands is not yet well defined in relation to the day of the fever. It seems certain, however, that it occurs within the first week; but it may be later. A case is described by Dr. Sankey, in the first volume of the *Pathological Society's Transactions*, in which dissection showed the growth in the glands as early as the fifth day. The bulging of the patch and the extent of intumescence vary considerably in different patches; and simultaneously with these conditions the mesenteric glands begin to increase in size. They, too, are supplied with an increased quantity of blood, and the increased tissue of the gland becomes unusually soft and elastic.

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III. *A subsidence of the general congestion, and of the generally turgid state of the mucous membrane, takes place after the gland-growth has been fully developed.* Nevertheless, the growth continues actively, and progresses rapidly till the patches of Peyer become so thick as to be elevated three or four lines above the surface of the mucous membrane. A beautiful vascular halo encircles them, stopping short at their margins; and a contracted border surrounds the margin of the patch, which gives it a sessile fungiform aspect, with an umbilicated-like depression on its surface. Growth is now confined within narrow limits, pressing on the muscular coat below and the mucous coat above. The patches assume various aspects as to colour; and, when vascular, they have an appearance which has acquired for them the description of being like "fleshy lumps;" their tawny grey colour showing through the peritoneum of the gut. *Varicose vessels abound in the vicinity*,—a fact of some importance in connection with the formation of thrombi, and which may lead to hepatic or

pulmonary embolism. The specific gravity of the mucous tissue of Peyer's patches is obviously changed by such increased growth, ranging from 1·032 to 1·044.

IV. *Softening of the contents of the tumid gland-cells* seems to be the next event in the series, and which would appear to be preliminary to one or other of the following results, namely:—

V. *Conditions under which the softened contents of the glands begin to be eliminated.* This elimination seems to take place in one or other, or in each of the three following ways; conveying out of the body by the intestinal discharges abundance of morbid material, presumed, with great probability, to contain the specific virus of the fever:—

1. *Elimination without ulceration—simply by the rupture of the hitherto closed gland-vesicles.* This is the usual and natural way in which the vesicles of Peyer's patches become open follicles in the course of their normal physiological existence. For many reasons I am induced to believe that this is the natural, the most common, and the most frequent mode by which the softened new growth in typhoid fever is got rid of—namely, by the escape of the softened contents of the glands through the rupture of the vesicles in the ordinary way. The vesicles then collapse, and assume the appearance of little pits, depressions, or follicles, and so give rise to that “reticulated indistinctly pitted surface” so often seen after all evidence of gland-structure has disappeared. This view is also consistent with the observation of Wedl, when he says that “the glands in question not unfrequently burst; and the capsules also may collapse, in consequence simply of absorption of their contents. Owing to one or other of these occurrences, the Peyerian patches acquire the well-known reticulated aspect, since the mucous membrane surrounding the individual capsules assumes the form of a projecting border; and entire patches present the appearance of a fine sieve” (*Pathological Histology*, p. 221). The ordinary peristaltic action of the intestines may assist this mode of elimination by rupture of the vesicles, if the softening is complete. One case I dissected at Scutari led me first to this conclusion. In this case a process of growth and elimination seemed to have gone on for at least one month previous to death; and the elimination of the material from the patches took place without ulceration. The man died suddenly from aneurism of the aorta. A lull in the febrile symptoms had led to his premature

discharge from hospital; and the day on which he suddenly died was to have witnessed his embarkation for England. Peyer's patches were in an extremely interesting condition. They were all large and obvious. In some parts of them the vesicles were greatly distended with the material of growth, in a milky-like condition; while other parts of the same patch were completely bare, and dotted over with minute points of black pigment. These parts were quite bare of all gland-structure, and had a reticulated appearance.

In corroboration of this view, it is to be observed, further, that Dr. Friedrich, of Dresden, considers the elimination of the deposit from the Peyerian patches in the typhoid fever of children, by the formation of sloughs and ulceration, as extremely rare. For the most part, only single follicles [vesicles?] in the glandular assemblage are infiltrated, and these, either from resorption of the infiltrated material, or more often from rupture of the follicle [vesicle] within the intestinal canal, *revert to a normal condition, without the formation of any cicatrix* (*Brit. and For. Med.-Chir. Review*, July, 1858, p. 162).

2. *Elimination by ulceration of the swollen gland-vesicles* in groups of various sizes, involving more or less of surrounding tissue in ulceration, and tending to induce perforation of the gut or peritonitis. Considering the severe nature of this lesion, and taking into account the fact that a large proportion of cases of typhoid fever recover, it seems to me that this is a mode of elimination which occurs much less frequently than the mode already described. Of course, it is the state most frequently met with after death; for ulceration of Peyer's patches is the characteristic "anatomical sign" of typhoid fever. The time of commencing ulceration of the mass appears to be about the ninth or tenth day (MURCHISON); and the softening which precedes ulceration is associated with a return of the violent congestion to the small intestines, when the veins especially are filled with dark-coloured viscid blood. The outbreak of the ulcers is always characterized by an aggravation of the original symptoms, after it may have been sanguinely supposed that convalescence had decidedly taken place. But in such deceptive convalescence the abnormal temperature is maintained, showing with absolute certainty that the fever is not at an end. The ulceration therefore is usually denoted (*a.*) by a re-accession of the febrile phenomena,

with or without diarrhoea; (b.) by abdominal pains and tenderness. Judging from *post-mortem* examinations, the ulceration seems to commence at the lowermost patches in the glands nearest to the cœcum, and the ileo-cœcal valve is often implicated in the destruction. The ulcers vary in number and in extent; and although there is a tendency to perforation of the gut in fatal cases, yet actual perforation is not common, and peritonitis may supervene without perforation having actually taken place. Various statements have been made concerning the tendency of typhoid ulcers to perforate the gut, and the frequent association of this lesion with peritonitis. Perforation is said to be rare in the northern parts of Europe (HUSS); but, from the records of Drs. Murchison and Bristowe, it appears to be a more frequent mode of fatal termination than has been commonly supposed in this country. Of fifty-five fatal cases, perforation occurred in eight (LOUIS); of fifteen fatal cases, perforation occurred in three (MURCHISON); of sixty-three fatal cases, perforation occurred in twelve (London Fever Hospital Records); of fifty-two fatal cases, perforation occurred in fifteen (BRISTOWE).

From these data it appears that perforation occurs in about one in five fatal cases; and it generally takes place through the ileum near the valve. *Post-mortem* examination often discloses vigorous attempts on the part of neighbouring structures to limit by union and adhesion the results of perforation, obviously indicating, in practice, the necessity of absolute rest throughout the disease.

The characters which distinguish the ulcers of typhoid fever from other ulcers of the intestines may be stated as follows:—(1.) They have their seat in the lower third of the small intestine, their number and size increasing towards the ileo-cœcal valve. (2.) They vary in diameter from a line to an inch and a half; but a number of ulcers may unite to form a mass of ulceration several inches in extent. Such extensive masses of ulceration occur close to the cœcum. (3.) Their form is elliptical, circular, or irregular—elliptical when they correspond to an entire Peyer's patch, circular when they correspond to a solitary gland, and irregular when they correspond to a portion of a Peyer's patch, or when several ulcers unite to form one. (4.) Elliptical ulcers are always opposite to the attachment of the mesentery. (5.) The ulcers never form a zone encircling the gut, as may sometimes be seen

Marking the Typhoid Ulcer

in the case of the tuberculous ulcer, but their long diameter corresponds to its longitudinal axis. (6.) Their margin is formed by a well-defined fringe of mucous membrane, detached from the submucous tissue, a line or more in width, and of a purple or slate-grey colour—an appearance best seen when the bowel is floated in water. (7.) After separation of the slough there is no thickening or induration of the edge of the ulcer, as in the case of the tuberculous ulcer. (8.) Their base is formed by a delicate layer of submucous tissue, or by the muscular coat, or occasionally by nothing more than peritoneum. (9.) There is no deposit of morbid tissue or new growth at the base of the ulcer; although sometimes fragments of yellow sloughs may be seen adhering to both the base and edges (MURCHISON, p. 547). The ulcers also are known to heal. Their cicatrices have been seen four, five, and thirty years after known attacks of typhoid fever (ROKITANSKY, BARRALLIER). And in cases where death occurs during a relapse, the cicatrices from the first attack may be found co-existing with the fresh growth in the vesicles, and with the recent ulcers of the relapse. As a rule, the reparative process does not commence till the end of the third week of the disease, and in one case, where the primary fever lasted three weeks, and where death occurred from complications about the fortieth day, all the ulcers in the ileum were cicatrized (MURCHISON).

Cicatrization commences by the growth of a thin, delicate, shining layer of new growth which covers the base of the ulcer, and is also attached to the basement membrane of the mucous coat. The fringe of mucous membrane becomes adherent to this new tissue, from the circumference towards the centre, until the healthy mucous membrane merges insensibly into the serous-looking lamina. The new film of membrane cannot at first be moved upon the subjacent coat, but after a time it becomes moveable, and according to Rokitansky, even becomes covered with villi (MURCHISON). There is no evidence of the vesicular gland-structure ever being restored. The resulting cicatrix is slightly depressed, firmer, less vascular, and smoother than the surrounding mucous membrane. The bowel appears thinner at this part when examined by transmitted light. The depressed spot is never surrounded by any puckering, nor does it ever cause any diminution in the calibre of the gut.

3. *Elimination of the typhoid growth by sphacelus of large*

masses of Peyer's patches. The whole gland-substance implicated is involved in the destruction. The cell-growth in the vesicles suddenly becomes so excessive that a condition is at last reached which is incompatible with the maintenance of life. The growth actually chokes itself; and the whole mass, or a great part of it, softens and dies. Such sphacelus has been known to happen as early as the twelfth day; but the process is generally more slow. A dirty yellow-brown slough forms, varying in thickness, and sometimes extending as deep into the substance of the gut as to expose its muscular layer on separation of the mass. There is a tendency to bleeding on separation of the sloughs; and such hæmorrhage occurs in about one-third of the fatal cases. The frequent repetition of such hæmorrhages during life has a marked influence in modifying the febrile phenomena. For example, in a case described by Dr. Parkes, in which the temperature was very carefully recorded three times daily, it was observed to fall *below* the standard of health on the fourteenth and fifteenth days slightly, on the sixteenth day to the extent of 4° below 98° , on the seventeenth day to the extent of 5° , on the twentieth day to the extent of 2° . These falls of temperature were all traceable to the influence of repeated hæmorrhages from the bowels. The occurrence of hæmorrhage is always a most alarming symptom, and is most frequent during the third and fourth weeks of the disease. It varies in amount from a mere stain to a large quantity of blood, sometimes discharged in clots, and generally of a red colour, in consequence not only of the rapidity with which it is passed out, but also, as Dr. Parkes has shown, in consequence of the alkaline re-action of the contents of the intestine. It may cause immediate death by syncope; or, by reducing the temperature and strength of the patient, he may sink exhausted, unable to cope with the disease. Whenever, therefore, blood appears in a case of typhoid fever, it is certain that the lesions of Peyer's patches are severe.

In addition to these three modes of elimination of the new growth from the intestinal glands, there are reasons for believing that it may be occasionally *re-absorbed*; unless such cases where resolution, independently of ulceration, commencing about the tenth day, may not be explained by the first method of elimination I have described.

VI. The mucous membrane of the intestines having existed

for several weeks in the state of irritation which has been described, and the catarrh being more or less excessive, *an atrophic condition of the intestine at last supervenes*. The mucous tubes become wasted, irregular in form and size, sometimes separated by an interstitial growth of a granular nature, their bulbous ends disappear, and the whole substance of the gut becomes so thin that it resembles a portion of thin paper rather than intestine.

209 The mesenteric glands are invariably enlarged. They begin to enlarge at the very commencement of the disease, and sometimes attain a very large size, and their stages of congestion, of swelling, and of subsidence, go on simultaneously with the similar changes in Peyer's patches.

The spleen is usually greatly enlarged, varying from five or six to fourteen ounces, with a specific gravity varying from 1052 to 1059. Its Malpighian sacculi (glandular) are also intumescent.

Pulmonary lesions occur (1.) as infiltrations, or (2.) as the consolidation of pneumonia, or (3.) as portions of lung which have become carnified.

(1) In the first-mentioned form of lesion the growth seems to commence in the terminal air-vesicles, ultimately assuming the form of a miliary deposit, with a semi-transparent gelatinous appearance. It is the irritation set up by this sudden growth which generally gives rise to pneumonic consolidation. Softening and friability of the pulmonary texture is thus a very constant *post-mortem* state in protracted cases of typhoid fever. Such lesions usually supervene during the later period of the fever, and when the ulcerations of the intestines are extremely spread (HUSS). In this respect only it differs from the consolidation of the lung to be described in typhus fever. This lesion has been also termed *non-granular consolidation*, dependent for its origin and development on a specific cause; and it may be observed not only in the course of *typhoid fever*, but in *measles*, *scarlet fever*, and *small-pox*. A portion of the lung in this condition has a mottled aspect. There are patches in it here and there, varying in size from a single lobule to half, or more than half, of a lobe, of a deep bluish-chocolate, violet, or purplish-slate colour, bounded by a well-defined angular margin, and crossed and mapped out into smaller patches by dull, opaque, whitish lines. These are seen to be thickened (into) lobular septa. Scattered in the midst

of the larger patches, one or more comparatively healthy lobules are frequently found, of a pale brightish-pink colour, contrasting strongly with the hue of the surrounding tissue. The pleura which covers the part may have a slight milky-like aspect (DR. JENNER). It is also extremely probable that much of these thoracic lesions in such cases may be due to the direct passage of fibrinous particles from the large veins surrounding the diseased intestinal glands (in the case of typhoid fever); for clots thus tend to form in the blood-vessels, near the site of irritation—they break up—the blood becomes contaminated, and the phenomena of *embolism* supervene. Such dangerous phenomena may be looked for about the fourteenth to the twenty-first day.

The tissue of the darker portions appears tougher than in health, presenting nearly a uniform section; there is no appearance of granules, and the part sinks in water. Dr. Jenner has injected such morbid lungs, and found that occasionally the centre of the lobule is really the point at which the diseased action is first set up. The development of the new material appears to be very deficient; molecular granular matter and delicate minute cell-forms compose its structure; and the specific gravity of the part is greatly increased (1.040 or more). Its colour is generally slate-grey or flesh-like; and the lesion is commonly limited by a vascular boundary, forming something like a distinct line of separation between comparatively healthy texture and local lesion.

Carnification occurs often in considerable portions of the lung (WALSHE). The general debility of the typhoid state seems to favour the occurrence of pulmonary collapse—a condition which must not be confounded with the hepatization of pneumonia. The tracheal glands, and those of the bronchial mucous membrane, are also affected.

There is generally a great tendency to ulceration of mucous membranes in typhoid cases,—ulceration of the pharynx occurs in about *one-third* of the cases; of the larynx and œsophagus in one out of every fifteen cases; and the mucous membrane of the colon becomes implicated in seven out of twenty cases. Louis found the colon affected by the second week in two out of fourteen cases, by the third week in six out of twenty-three cases, by the fourth week in four out of fifteen cases, and between the fifth and tenth week in one out of two cases.

There is also a tendency to pericæcal abscess, preceded by the phenomena of the morbid state known by the name of *perityphlitis*, or inflammation of the areolar connective tissue round the *caput cæcum*.

Growth of Tubercle during Typhoid Fever.—There is still another pulmonary condition which frequently occurs in *typhoid* fever, and which may either complicate the progress of the case or come on subsequently to the fever. It is the development of tubercle. Usually when recovery takes place from *typhoid* fever it is complete; but in some cases, especially where there is hereditary predisposition, an impetus or tendency seems to be given to the development of tubercles in the lungs. If the physical signs of bronchitis continue beyond the thirtieth day, or fourth week, combined with hurried and difficult breathing, and with the signs and symptoms of great irritation of the lungs, then there are good grounds for suspecting that the deposition of tubercle has commenced in the lungs. Dr. Stokes gives two sets of cases in which this deposit takes place. In one set a great quantity of tuberculous matter seems to be formed during the existence of the fever; and although, sometimes, such an occurrence may not have been suspected, yet the expectoration of pulmonary calculi, at periods of different duration after the convalescence, furnish strong proofs that such a lesion had taken place. In other cases, again, the cure may be effected through absorption, or by suppuration of the minute tuberculous points over the mucous surface of the bronchia. A doubtful convalescence, a quick pulse, and a hectic state, suggest such a state of things, especially when combined with persistent bronchitis.

Erysipelas, phlebitis, parotitis, and such-like local inflammations, are not uncommon in typhoid fever. Such lesions may be excited by cold simply; but the absorption into the blood of putrid substances, from the ulcerating patches of Peyer or other diseased parts, may be usually, and probably correctly, considered to be the cause of most of the secondary inflammations already noticed to occur in typhoid, enteric, or intestinal fever. Dr. Parkes considers it probable, however, that deficient urinary excretion may have a share in their production (Parkes *On the Urine*, p. 254).

Such are the more obvious secondary affections which may develop themselves during the progress of *typhoid* or *enteric* fever,

and the derangements which these give rise to constitute new phenomena in its course. In some severe cases, however, the fever may destroy the patient in a few days, without leaving a trace of organic lesion in any part of the body.

These secondary affections just noticed all arise after the fever has existed some time; and it appears now to be pretty well established that the intestinal lesion at least is a special growth, which, in cases of recovery, follows first a progressive or developmental course, and afterwards retrogrades; just as in variola we first observe the development and maturation of the pustule, and subsequently its disappearance. The same may be said of the other local lesions in typhoid fever, although the existence of a special growth is not yet so fully established in the case of the thoracic and cerebral lesions, or in the parenchymatous, as compared with the mucous structures of the intestine; still, it is believed by some that an action more or less analogous to that which occurs in the *glands of Peyer* and the *minute solitary closed vesicles of the ileum*, occurs also in all the secondary lesions of typhoid fever in other parts (DR. STOKES). Specific characters of the elements composing the growth cannot be shown to the eye even by microscopic examination. There is nothing in it to distinguish it from other elementary morbid products which are deficient in the power of organization. Dr. Stokes gives the best illustration of its vital specific attributes, in the absence of any *physical* specific character. He says, if two specimens of pus be taken, one from a pustule of variola, the other from an ordinary ulcer, although they may appear similar, they have separate and different *vital* characters. So has the poison of typhoid fever a specific *vital* attribute peculiar to itself.

The nature of this so-called "typhoid deposit" has been the subject of much discussion. It is a new growth rather than a deposit; and is, in the first instance, confined to the gland-elements, and seems really to consist in a directly continuous development of the pre-existing cell or germinal elements of the diseased glands. Eventually it pervades the submucous areolar tissue as a yellowish-white substance, deposited or infiltrated in a layer beneath the gland-tissue. Professor John Goodsir, in his descriptions of the morbid anatomy of the cases he dissected at Anstruther, in Fifeshire, was the first to point out that the new growth was in the first instance confined to the interior of

the closed vesicles, which became much distended thereby. They ultimately burst, and discharge their contents either into the cavity of the intestine, or into the submucous tissue, if the vesicles rupture at the base, as in the severe and unfavourable cases; and if the vesicle is completely destroyed and falls out, or if many of them do so, a number of little pits are left, which correspond to the sites of the vesicles.

The new growth has no specific structure to distinguish it from other morbid growths (WEDL, VIRCHOW, and others); and although a specific "typhous cell" has been described and figured by Gruby, Vogel, Bennett, and others, its existence is not proven.

The development of the new growth in the glands begins like a simple hypertrophy of the gland-cells. Nuclei and cells exist in great abundance, which afterwards degenerate into the abnormal diseased product of the typhoid masses. No forms arise capable of sustained existence; but a directly continuous development from pre-existing cell or germinal elements of the glands, the follicles, and the connective tissue, furnishes the material of the mass (VIRCHOW). An increase in the colourless corpuscles takes place in the blood, and deposit of pigment in the ganglia of the sympathetic nerve-system (VIRCHOW). When the gland-vesicles burst, the exuberance of new growth gives a fungating appearance to the part; and when the rose-red tumor is cut into, a milk-like turbid juice exudes; and in this juice many new-formed elements may be seen, consisting of cells mostly oval or angular, with single eccentric nuclei—sometimes with many nuclei. The cell-contents are finely granular, and fat-globules may conceal the nucleus. The growth must be examined before ulceration commences; for, as softening advances, a viscid fluid with a bloody tinge, containing fine molecular elements, is all that remains, with decaying blood and blood-crystals.

Circumstances under which Death may ensue in Cases of Typhoid Fever:—

1. By poisoning of the blood generally, as indicated by many symptoms which typhoid fever has in common with typhus fever, cholera, small-pox, dysentery, scarlet fever, diphtheria, ichorrhæmia. The intensity of the fever (measured by the thermometer) is generally great in those cases, and the fatal event occurs either at a very early period of the fever, associated with cerebral congestion, or it may occur later,

*Average Mortality 1 - 5 to 6
Spring most fatal period.*

when it may be supposed that the danger is past. This is sometimes termed the secondary poisoning of the blood (septicæmia), and is most likely due to the ulcerated intestines, with the bowels perhaps on the verge of perforation. The pulse becomes rapid and small; cold, clammy sweats appear; and the body begins, even in life, to exhale a putrid odour. In cases where the blood is so gravely implicated, gas has been observed to become developed during life, and has been detected in the veins at the root of the neck for some minutes before death (CLOSS, FRANK, and JEFFREY MARSTON in *Med. Times*, Feb. 7, 1857).

2. By implication of excretory organs at an early period—for example, the kidney, as denoted by albuminuria, or by bloody urine—conditions which tend to aggravate the blood poisoning.

3. By congestions of important organs—for example, the lungs and the brain, in consequence of poisoned blood; and which congestions are still further brought about by the circulation, in the blood-vessels, of putrid juices, or of the substance of fibrinous debris of clots in a granular condition, having formed as plugs in the varicose veins surrounding the sloughs and ulcers of the intestines.

4. By hæmorrhage from the bowels during the separation of the gland-sloughs.

5. By exhaustion from profuse diarrhoea in cases where the catarrh of the mucous membrane has been excessive.

6. By peritonitis, with or without perforation of the intestines. There are two periods in the course of the fever when perforation is apt to take place. The first period is during the separation of the sloughs, about the end of the second and throughout the third week. The second period is during protracted convalescence, with atrophy of the intestine already described, and when the ulcers are in a weak atonic state, the result of intense protracted fever and profuse catarrh.

7. By peritonitis subsequent on suppuration of the large mesenteric glands, and rupture of their enclosing capsule (JENNER); or from the bursting of softened new growth from the spleen into the peritoneum (ROBERTSON, JENNER); or from ulceration of the gall-bladder. The average mortality among cases of typhoid fever appears to be about 1 in $5\frac{1}{2}$ to 1 in 6. It is considerably less in Autumn than in Spring; and is least of all in Winter. It

tends to be greater among males than females; and the average age of fatal cases appears to be about 23·5. The mortality increases to a small extent as life advances. The disease in certain places seems never to be absent, and is invariably most prevalent during Autumn, at the time that diarrhoea is most common; and it has been observed to be especially prevalent in seasons remarkable for their high temperature (MURCHISON).

Origin and Propagation of Typhoid Fever.—It is now about thirty years since M. Bretonneau related to the French Academy of Medicine a series of cases in which the communication of this disease from person to person, and its modes of propagation in this way, were so evident as to admit of no reasonable doubt. Nevertheless, the conclusion arrived at has not been generally accepted by the profession, so that the communicability of typhoid fever has not met with general belief. “In so vital a question” (writes Dr. Budd, whose views, as I have attempted to put them here, seem especially deserving of attention), “it is, I need scarcely say, of the highest importance that the actual truth should be generally known.” And Dr. Watson, whose authority is undisputed, very justly remarks that, “If this fever be really contagious, it is not only erroneous but dangerous to hold the contrary opinion.” “To what extent it is dangerous,” continues Dr. Budd, “may be best measured by the fact, that in this country alone 20,000 persons die annually of this fever, and 140,000 more are laid prostrate by it.” Thus, vast is the field for the operation of preventive measures. And when the discovery and success of such measures must depend in a great degree on our insight into the real mode of propagation, it is at once seen what importance the question assumes. Dr. Budd has, I think, clearly demonstrated the following facts:—

1. That typhoid fever shows a decided tendency to spread through a household, a school, a barrack, or a village, when it has been once introduced. The introduction and propagation of the fever in the Clergy Orphan School at St. John’s Wood (*Lancet*, 15th Nov., 1856) is an instance in point. The first case was imported, and the illness began ten days after arrival; within three weeks four more cases occurred; and then *nineteen* cases were simultaneously affected within thirty-six hours. The fever which prevailed in the Military School of La Flèche, in France, in 1826, is a no less instructive example. The outbreak commenced

first with a few scattered cases, and the disorder spreading, the school was broken up earlier than usual, and the pupils sent home. But before this disruption could be carried into effect, sixty of the number were seized with typhoid fever. Twenty-nine others carried the seeds of the fever with them, and were laid up by it at their own homes. Of these twenty-nine, it was ascertained that *as many as eight communicated the disorder to persons who were engaged in attendance upon them.*

In further illustration of the doctrine of communicability, Dr. Budd cites, with minute details, numerous examples in the village of "North Tawton," in which typhoid fever having once appeared in a household, it extended itself to one or more members of the family before it finally died away. During the prevalence of the fever in this village, it also so happened that three persons left the place after they had become diseased, and each of the three persons communicated the same disease to one or more of the persons by whom they were surrounded in the new neighbourhood whither they went. While two of these men remained in the village of "North Tawton," they both lodged in a court having a single and a common privy in it, and next door to a house where typhoid fever was. In due course of time and events both took the disease. The third man was a friend who came to see one of the two men already sick. He assisted to raise his sick friend in bed, and while so employed was quite overpowered by the smell from the sick man's body. The sense of this pestilent smell harassed him for days. He felt very unwell from that time; and on the *tenth* day from the date of the event just noticed, he was seized with shivering, followed by the complete expression of an attack of typhoid fever, which was of long duration; and before he became convalescent two of his children were laid up with the same disease, as well as a brother, who lived at some distance, but who repeatedly visited him during his illness. Except in the houses of these men no fever existed in that part of the country. Further most interesting and conclusive examples are given in Dr. Budd's admirable papers published in the *Lancet* of 9th July, 1859, which prove beyond question that typhoid (intestinal) fever is a most readily communicable disease. The facility for propagation, however, seems to be modified under particular circumstances of season, place, and habits of life; in other words, the propagation of the disease requires some special

conditions, which may be said of the whole class of communicable diseases.

2. The disease having once occurred, the patient is protected from a second attack. The specific nature of the disease is thus also established, for the fever not only propagates itself, but propagates no other kind of fever—one case following another with the same constancy of specific type that small-pox follows small-pox or measles succeeds to measles.

3. There seems also to be a definite period in which the poison is latent after being communicated—a period of incubation, during which a definite interval elapses before the development of the fever begins. This period, according to Dr. Budd's experience, seems to be from about a week to ten or fourteen days. *The living human body, therefore, is the soil in which this specific poison breeds and multiplies; and that most specific of processes which constitutes the fever itself is the process by which the multiplication is effected.*

4. Like all other diseases of its kind, its origin is unknown; and the first case in the series of each of these outbreaks mentioned may either have been casual and imported, or it may have been due to a rekindling of some dormant germ left from a former similar attack.

W3 || 5. The virulent part of the specific poison by which the disease is communicated is doubtless contained in the diarrhoeal discharges which issue from the diseased and *exanthematous* bowel. These discharges drying up, the germs of disease are thus preserved as effectually as the crusts of small-pox preserve the virus of that disease. If, therefore, through atmospheric or other agencies, these germs obtain access to the living body, diarrhoea is brought about in the usual course of events, and the commencement of the disease thus communicated takes place. The discharges from the bowels of the person so infected, which are at once copious, numerous, and liquid, are thrown into the water-closet or the privy, and the drains, or systems of drains, become at once saturated with the specific poison of the disease in its most concentrated and virulent form.

Regarding, therefore, the drain, or system of drains, as a channel directly continuous with the diseased intestine of the infected person, the specific virus of typhoid fever may be propagated amongst healthy persons in one of three ways—namely,

(1.) By percolation through the soil into the wells which supply drinking-water to the inhabitants; (2.) By issuing through defects in the sewers into the air of the inhabited area; or, (3.) By exhalation through the aperture of small, ill-trapped water-closets or privies, which are at once the receptacles of the discharges from the sick and the daily resort of the healthy.

When the specific poison thus issues into the air, the *atmosphere generated is immeasurably* more likely to communicate the disease than that which immediately surrounds the fever patients. "There is reason to believe, however, that the duration of the period of incubation varies considerably, partly with the nature of the medium through which the specific poison finds admission to the living body, partly by reason of the conditions it meets with there, and still more so *in virtue of the greater or less intensity of the state of change* in which the poison itself may be at the moment of its reception." Hence the simultaneous seizure of a large number of persons within a definite interval after the occurrence of a single case points to some one or other of these modes of propagation. Such modes of propagation are thus the exact counterpart of what has been oftentimes observed (especially in schools) in the case of measles, scarlet fever, and, in former times, of small-pox also (DR. W. BUDD).

Preventive Measures, or Measures for Checking the Spread of Typhoid Fever.—The measures about to be specified have been made public through the writings of Dr. William Budd on this subject; and, *provided they are thoroughly and efficiently carried out*, it is believed that the recurrence of typhoid fever may be entirely prevented.

To enable us to judge of the extent of the infection to be destroyed, there are two elements to be taken into account,—*First*, The amount and duration of the intestinal discharge in each case; and, *Second*, The number of cases actually occurring. With regard to the first, Louis has found that *the average duration of the alvine flux in cases of typhoid fever is fifteen days in mild cases and twenty-six days in severe cases*. With regard to the second point—namely, the number of cases occurring—the Reports of the Registrar-General show that at least 100,000 to 150,000 cases of typhoid fever occur annually in England alone. In other words, "*every year in England more than 100,000 human intestines, diseased in the way already described, continue*

each, for the space of a fortnight or thereabouts, to discharge upon the ground floods of liquid charged with matters on which the specific poison of a communicable disease has set its most specific mark” (BUDD).

The measures recommended for preventing the spread of this fever are founded on the power of chemical agents to destroy absolutely the material which contains or carries the specific virus of such communicable diseases. Assuming it, therefore, to be certain that the intestinal discharges in typhoid fever are the media of propagating the disease, it is no less certain that, by SUBJECTING THE DISCHARGES ON THEIR ISSUE FROM THE BODY TO THE ACTION OF POWERFUL DECOMPOSING CHEMICAL AGENTS, THEY MAY BE ENTIRELY DESTROYED OR DEPRIVED OF THEIR SPECIFIC VIRUS. He suggests the following details of procedure:—

1. All discharges from the fever patient should be received on their issue from the body into vessels containing a concentrated solution of chloride of zinc.

2. Two ounces of a caustic solution of chloride of zinc should be put in the night-stool on each occasion before it is used by the fever patient.

3. All tainted bed or body linen should, immediately on its removal, be placed in water strongly impregnated with the same agent.

4. The water-closet should be flooded several times a day with a strong solution of chloride of zinc; and some chloride of lime should be also placed there, to serve as a source of chlorine in the gaseous form.

5. So long as fever lasts, the water-closet should be used exclusively as a receptacle for the discharges from the sick.

The Privy Council have now made the principle of this method an integral part of their “general memorandum on proceedings advisable to be taken in places attacked or threatened by epidemic disease,” and which is given *in extenso* at page 228 of this volume.

Vague and untenable notions have been gathering round this subject, particularly in relation to the propagation of typhoid fever; and if once a disease of this kind is decidedly proved to be the result of a specific poison, and to be propagated in the way just described, “we cannot help entertaining a doubt,” says Dr. Watson, “whether the disorder in question really ever has any

other cause." Nevertheless, other causes are assigned to typhoid fever; which by some is even looked upon as a disease developed out of external conditions alone. In reasoning on the subject, it must be remembered at the outset that the dissemination of typhoid fever by a *specific poison* implies precisely what it implies in small-pox; and that it is provided for in the same way—namely, by the multiplication of a *specific poison* in the living but diseased body. Each of these specific poisons, (and, as we have already seen, they are numerous) thus multiplies in the same way, and in the same remarkable medium, out of the same living organisms of the human frame; yet each of these several poisons sets up a series of changes which always issues in the reproduction of its own specific kind of disease, and no other. Small-pox gives rise to small-pox, scarlet fever to scarlet fever, measles to measles, and so on. Herein lies their *specificity*. Such being the doctrine attempted to be maintained in these pages, the theory of the spontaneous origin of typhoid fever, or of any other specific disease, must be in the same relative position as when it seeks to explain by such a principle the propagation of plants and animals. These—namely, plants and animals—likewise at least two diseases—namely, *syphilis* and *small-pox*—are certainly now known to propagate only by the law of continuous succession, whatever may have been their primary source. But the hypothesis of spontaneous origin and indefinite propagation of typhoid fever has assumed a definite form of expression in the doctrine which attempts to teach that typhoid fever is often actually caused by the products of common putrefaction—a doctrine which has been cleverly embodied in the nomenclature of the subject by Dr. Murchison; and thus a degree of precision and permanence has been given to the opinions he has so ably advocated in a volume on the *Continued Fevers of Great Britain*—a volume which is unsurpassed for its erudition and its practical importance.

The term "pythogenetic fever," or fever "born of putrescence," is the name by which Dr. Murchison at once designates typhoid fever and theoretically implies its origin. He has thus rashly committed Science to a hypothesis of a highly doubtful nature. Without, however, doubting the fact that animal and vegetable substances in some states of decomposition have the power of inducing ill-health; and that there is now acknowledged to be a connection between putrid states of the air and the prevalence of

pyæmia, erysipelas, puerperal fever, and cholera, yet there does not seem to be sufficient evidence to show that any of these causes can *produce* a disease which is of so specific a nature as to be maintained and propagated by a specific poison generated in the body alone. Undoubtedly, the state of ill-health induced by the decomposing material of night-soil and the like, does produce *a state of the system favourable to the development*, not alone of typhoid fever, but of many other specific diseases, such as cholera, dysentery, yellow fever, and the like. This predisposition to such diseases seems to be exactly analogous to the preparation of a soil for seed. Dr. Carpenter, also, long ago showed physiologically what observation has since confirmed—namely, that decaying animal material, especially night-soil, seems to be for some poisons (*e. g.*, cholera and yellow fever) great centres or foci, where the specific germs or poisons are able to multiply; and for the propagation of which “foulness of medium is indispensable.” An interesting question for inquiry is thus opened up as to whether the germs of typhoid fever, cholera, and the like, could be made experimentally to grow or increase upon or about organic matter, just as the germs or spores of many fungi are induced to grow in collections of manure (DR. LANKESTER). In such collections on the earth’s surface there is reason to believe that germs of diseases like cholera, and typhoid fever, and yellow fever, may find a resting-place—that thus they are always extant somewhere—although it may be only now and then, when season and other conditions conspire, that they display their full power as epidemic diseases. As such, they seem to occur every now and then as “mysterious cycles,” the existence of which we admit, but do not understand. The experiments of Dr. Barker on cess-pool air prove that long inhalation of an atmosphere charged with the gases evolved from decomposing organic matter is capable of producing a series of symptoms of the following character—namely, increased heat of skin, thirst, irregular and feeble muscular contractions, and diarrhoea. These symptoms continue so long as the person is exposed to the influence of the foul air; but when the cause is removed, there is no continuance of symptoms, no recurrence nor remittency, but a tendency to recovery. No communicable disorder is induced. The poison of the foul air acts for the time as chloroform might act, and so soon as removed, recovery progresses.

The history of typhoid fever, whose leading features have been

described in the previous pages, is wholly inexplicable upon the "pythogenetic theory" of Dr. Murchison. On the contrary, it is emphatically the history of a specific disease generating a specific poison, and propagating itself by it:—

"*Mutatis mutandis*," writes Dr. Budd, "it is the history of *small-pox*, it is the history of *scarlet fever*, it is the history of malignant cholera. In all these specific contagions we meet with these same alternations of slumber and activity; of wide-spread prevalence in one place, while other places hard by remain free; and finally, with the same successive invasion of neighbouring places, in such wise that the reigning disorder—be it small-pox, measles, scarlet fever, intestinal fever, or malignant cholera—often only begins to prevail in the new locality when it has already died out in the old.

"It is, in fact, in a general survey of this kind that we get the clearest view of the thread which really connects all these circumstances. There is plainly but one thing constant; that is to say, a specific morbid cause—a cause which is neither a permanent product of the soil, or air, or of particular seasons, but which is susceptible of transmission from place to place; which breeds as it goes, and then again dies out or becomes dormant, without leaving any sign to mark its track.

"There is only one thing of which these can be the characteristics; and that is the specific poison which is bred of the disease, and by which the disease propagates, and which, in common with the other specific poisons perpetuated by the same law, possesses all these properties.

"Thus, when we come to scrutinize closely this course of the fever, even in these broad relations, we are again brought to recognize that which we have already proved by direct evidence—namely, its essentially contagious nature. This is the master fact in its history—the fact which governs all the rest."

But whatever may be the view theoretically adopted regarding either the origin or the propagation of typhoid fever, it is satisfactory to know that, practically, medical officers can employ preventive measures which (to use the words of Dr. Lankester) "will cover the issues of both theories." These measures have been minutely described; and if they are universally carried into effect, it is not too much to expect that this fever might perhaps soon become extinct. At all events, with the facts before us, it is unwarrantable to permit the great bulk of what escapes from the diseased intestine of typhoid fever patients to be let loose upon society, into the cess-pool or sewer, or on the dung-heaps,

in full possession of all their deadly power, without being first destroyed in the way that has been recommended.

"The grand fact is clear," writes Dr. Parkes, "that the occurrence of typhoid fever points unequivocally to defective removal of excreta, and that it is a disease altogether and easily preventible." Typhoid fever ought therefore soon to disappear from every return of disease, whether in military or in civil life.

Treatment of Enteric or Typhoid Fever.—The chief indications of treatment are to reduce temperature and subdue vascular excitement, if these be in excess; to restrain and moderate, but not to suppress or check, the diarrhoea; to stimulate the nervous system when necessary; to obtain a free action of the kidneys; and to influence the elimination of the morbid growth from the intestinal glands.

To accomplish the first of these indications the use of *digitalis* has been especially recommended by Wunderlich. He considers that it decidedly mitigates the febrile symptoms which are present in severe cases at the time when the ulcers begin to heal, and which often impede or prevent recovery. He advocates its use in the severe forms of the fever only, especially at a time when most danger is to be apprehended from the violence of the fever in the second week, when the evening temperature is at its highest (105° to 108° Fahr.), and when the remissions in the morning are slight; when the pulse is frequent, 110 to 120 or more. In mild cases it is superfluous. He finds that in the form of *infusion* it is easily absorbed by the intestines of patients suffering from fever; and, if given in a suitable dose, has most marked effects in subduing the rate of pulsation, and in reducing animal heat. Large doses of the infusion should be given without interruption until the full effect has been obtained,—

An infusion of fifteen or twenty grains of *digitalis* in eight or ten ounces of boiling distilled water may be consumed in twenty-four hours by adult patients.

It acts more rapidly on animal temperature than on the heart. For the first few days after its use the decrease of temperature is rather slight, but may afterwards become considerable; and after it has been much diminished and again rises, it never attains its former excessive height. The full effects of the medicine are known to be brought about if the temperature is

reduced to 2° or 3° Fahr. in the evening; and the action of the remedy does not continue beyond one day after its use has been discontinued. The diminution of the pulse is slight at first, and occurs in some cases on the second day after the remedy, but mostly on the third day, or even later; and on the fourth or fifth day after the medicine has been commenced, the rate of pulsation may be diminished by thirty to forty or fifty beats within from twelve to thirty-six hours. The pulse may continue to fall even below its normal velocity, and this reduction of pulsation may last for several weeks in succession. If the velocity of the pulse should decrease rapidly, the use of the digitalis must be discontinued at once (*Arch. der Heilk.*, 1862, p. 116).

Cold and tepid sponging, or the cold affusion, are remedies which Dr. Murchison considers deserving of further trial for reducing the pulse and temperature. The oppressive headache may be greatly relieved by cold to the head; and in all severe cases the hair should be shaved off. (*Watson*)

To restrain excessive diarrhœa, *lime water mixed with milk* in equal parts, and taken as a drink, is found to be beneficial, agreeable, refreshing, and nourishing. It is clear that the diarrhœa *ought not* to be altogether checked. Professor Gairdner, of Glasgow, is in favour of the French practice of giving saline laxatives rather than astringents; also of the diligent use of enemata to unload the bowels from below, where anything like abdominal distension has occurred. These enemata may be simply of warm water, to which a little aniseed is added; or the asafoetida enema may be given. On the propriety of restraining *excessive* diarrhœa there can be no question. Dr. Huss, of Stockholm, is of opinion that the diarrhœa during the first stage ought not to be arrested, but abated and mitigated only *if excessive*. If it is suddenly arrested, meteorism is produced, and pains in the intestines; or vomiting may supervene, with cerebral symptoms, and the febrile phenomena are increased. The diarrhœa is too copious or excessive if the evacuations exceed four or five a day, being of considerable quantity, and fluid. Such evacuations weaken the patient rapidly, and should be mitigated by mucilaginous drinks, such as rice-water, infusion of lintseed, decoction of *althæa officinalis*, or *ipecacuanha* in small and repeated doses. This latter remedy retards the peristaltic action of the intestines, and lessens the secretion from the mucous

membrane. The dose must be regulated so as to avoid vomiting; and the feeling of nausea which is apt to follow the first dose soon disappears with continued use.

Dr. Murchison, on the other hand, agrees with the late Dr. Todd, who writes as follows:—"Restraining diarrhœa and hæmorrhage in typhoid fever, and when you have fairly locked up the bowels, keep them so. Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation." Dr. Huss and Dr. Murchison speak highly of the benefits to be derived from the mineral acids—hydrochloric and sulphuric especially. From fifteen to thirty minims of the dilute acids may be given every three or four hours; and with each dose Dr. Murchison recommends half a grain of quinine, as in the following prescription for an adult:—

R. Acid. Sulph. dil., vel Acid. Hydrochlor. dil., ℥ xx. ad. xxx.

Quiniæ Sulph., gr. $\frac{1}{4}$ ad. gr. i.

Syrup Aurantii, ℥ss.

Aquæ Carui, ad. ℥i.

Fiat haustus, 3â vel 4â, horâ sumendus (MURCHISON).

He is of opinion that if there be more than two motions in the twenty-four hours, with marked prostration, that astringents should be had recourse to. A starch enema, containing from ten to twenty drops of laudanum, should be administered towards evening, and recourse may also be had to the following draught:—

R. Acid. Sulph. Aromat., ℥ xxx.

Liq. Opii. Sedativ. (Battley), ℥ iii.

Aq. Menth. Pip., ℥i.; *misce*.

Fiat haustus, 4tâ vel 6tâ, quaque horâ sumendus (MURCHISON).

If the mineral acids are not tolerated by the stomach, acetate of lead is worthy of trial, in doses of two or three grains in solution every four or six hours, with or without an eighth of a grain of morphia (MURCHISON).

Alum dissolved in gum, to the amount of twenty-four grains in a day, which may be increased to one drachm, is best given in the form of *alum whey*, prepared by adding one drachm of alum to a pint of boiling milk, and then straining. Two ounces may be given after each motion of the bowels (FOUQUIER, MURCHISON).

A seidlitz powder may sometimes check excessive diarrhœa, by altering the secretions, and is especially beneficial if there be much meteorism (TROUSSEAU, MURCHISON).

The question, therefore, of checking excessive, or encouraging moderate diarrhoea is one which requires the careful study of each particular case; and my experience in the treatment of typhoid fever leads me to recommend rather the restraining of excessive diarrhoea than either encouraging the action of the bowels or locking them up for days. The French practice of giving saline laxatives is not a safe one; and if the action of the bowels is deemed desirable, enemata will be found sufficient. Nitrate of silver, in doses of one to three grains, made into a pill, and taken every six or eight hours; or sulphate of copper, in doses of a quarter of a grain similarly given, are remedies most useful in the diarrhoea connected with atonic ulcers, after the fourth week of the disease (BELL, MURCHISON).

If meteorism or tympanitis prevails, from the accumulation of air in the colon, it may sometimes be relieved by the passage of a long stomach-pump tube by the anus, as far up the colon as it can be made to go easily. (Sanner)

With regard to stimulation of the nervous system, it is not decided how far alcohol is beneficial. The fever eventually is attended with much exhaustion, and is often protracted, yet typhoid fever does not seem to bear stimulation so well as typhus. The tendency to prostration is the only indication for its use; but stimulation must not be persevered in if the pulse is quickened by its use, the hectic flush made more manifest, the tongue made drier, or if delirium supervene. Food and sustenance are the real preventives of delirium, and the best stimulants to the nervous system when necessary. With regard to the maintenance of free action from the kidneys, it is to be observed that so long as the excretion of urea and uric acid is abundant, no diuretics are necessary; but whenever the amount of the solids falls greatly (which can be known at once with sufficient accuracy by a comparison of the urine passed in twenty-four hours with the specific gravity), means must be taken at once to increase, if possible, the urinary elimination. The warm bath, with repeated small doses of the alkaline carbonates, or of the nitrate or bitartrate of potash, will often effect an increased elimination (PARKES, l. c.)

To accomplish the last indication—namely, to influence the elimination from the intestinal glands, by direct local action on the intestinal membrane—Wunderlich especially advocates the

use of *calomel*, if it can be given before the ninth or tenth day. Dr. Parkes is also of opinion that it is extremely useful at this period. The late Dr. Anthony Todd Thomson used to give it; and, from the observation of many cases under the care of this physician, as well as from his own experience, Dr. Parkes considers that *calomel* is a medicine to be strongly recommended in typhoid fever. But it must not be given later than the tenth or eleventh day, and at no time in large doses.

One or two grains twice a day is enough, although Wunderlich gives one to five grains twice daily; but five grains is considered by Dr. Parkes to be too large a dose.

Dr. Wood, of Philadelphia, bears testimony also to the benefit to be derived from mercury about the seventh or ninth day of the fever. He believes "it tends in some degree to arrest the progress of the disease in the glands of Peyer, and to promote resolution of the inflamed patches. He prefers minute doses of the blue pill mass—a grain every two hours—till the mouth is slightly affected, associated with small doses of *ippecacuanha*, when the stomach is not irritable. The beneficial effect of this combination is shown by the tongue becoming moist, the skin relaxed, and the symptoms generally being ameliorated. Dr. Wood recommends twelve grains of *blue pill mass* to be combined with two grains of *ippecacuanha powder*, with two grains of *opium powder*; and the whole being divided into twelve pills, one may be taken every hour, or every hour and a half, or every two hours (*Practice of Medicine*, vol. i., p. 345, 4th edition).

Calomel is, however, contra-indicated if the diarrhoea is excessive, or if there should be excessive pains in the bowels, with early and violent meteorism. It is also not proper to be given if the condition of the patient is anæmic, or if there is a decided hæmorrhagic diathesis. It is most useful as a restorative of the intestinal functions in cases where the tongue is dry and coated, where thirst is absent, and when the urine is cloudy and of low specific gravity. If the first dose is vomited, the administration must be repeated. Calomel has no direct effect on the pulsation or respiration, nor on the cerebral functions; but its beneficial influence is very decidedly appreciable by the modifications of temperature which it induces, and which have been already noticed at page 392.

No general rules can be laid down to guide the treatment of

the intercurrent phenomena or accidents of the disease. Combinations of remedies must be adapted to correct the several functions which may be simultaneously deranged, so that treatment must be varied according to the functions mainly implicated or suspended, and to the degree of their affection.

Abdominal pains and meteorism may be relieved by mustard poultices, or turpentine *stupes* may be applied, followed by simple hot water fomentations. After these remedies have been used, cold water compresses over the abdomen tend to lessen the tension and the gurgling in the intestines, and to diminish the tenderness on pressure. They counteract the inclination to meteorism, and lessen excessive diarrhoea. Dr. Huss believes also that the ulcerations in the ileum are prevented from spreading; and that perforations of the intestine have been of much rarer occurrence since he commenced to use these compresses. The compress, after being soaked in warm, but not hot water, is well wrung, and applied so as to cover the whole abdomen; and it must be changed two or three times a day, according to its tendency to dry. The compress is composed of four to eight double folds of coarse linen, and is to be laid over the whole abdomen, and afterwards overlaid with a cover of oiled skin or india rubber stuff, to prevent too rapid evaporation. The compress should fit as closely as possible, and care must be taken that it be not displaced, otherwise air enters between the skin and the compress, so that cold, instead of a moist heat, is produced. These compresses may remain untouched as long as they are moist and warm; and they may be employed on the chest as well as on the abdomen, should capillary catarrh or pneumonia supervene.

Hæmorrhage from the bowels, partly fluid and partly in clots, evacuated with the stools, is a symptom of grave import; and the urgent aim of treatment is to arrest the bleeding. Huss found the *sugar of lead* most serviceable, administered by the mouth, and also by clysters. By the mouth, two grains may be given every half-hour, and even every quarter of an hour; and it is best given dissolved. Twenty-four grains of *crystallized acetate of lead* being dissolved in one drachm of *dilute acetic acid*, to which six ounces of *distilled water* is added, a table-spoonful of this solution may be given every half-hour. At the same time a clyster may be administered, composed as follows:—Ten to fifteen grains of *acetate of lead* are to be dissolved in four ounces of

distilled water (warm), to which twenty or thirty drops of tincture of opium may be added; and the administration of such a clyster may be repeated in four or six hours if required. Pieces of ice may also be swallowed now and then; and even crushed ice may be applied, enclosed in a bladder, over the abdomen.

If bleeding from the nose is not arrested by the use of vinegar and cold water injected up the nostrils, nor by the use of cold water compresses applied to the nose, plugging by the posterior nares must be had recourse to.

Turpentine is a most valuable remedy in hæmorrhages, and in the stage of ulceration. It was originally recommended by Dr. Graves, and is highly spoken of by Drs. Huss, Wood, and Murchison. It may be given in all cases where the tongue is dry, and when, "instead of cleaning gradually from the edges and tip, it often parts with its fur quickly, and in large flakes—generally, first, from the middle or back part of the surface, which is smooth and glossy, as if deprived of its papillæ. There is also generally an increase of the tympanitis, and the ulceration of the ileum seems to be attended with great dryness of the tongue." Under these circumstances Dr. Wood gives the oil of turpentine in doses of five to twenty drops every hour, or every two hours. It is best administered in an emulsion with gum arabic, loaf sugar, water (WOOD), or in an emulsion with the yolk of an egg and honey or mucilage (HUSS). Amelioration of the symptoms may be observed in twenty-four or forty-eight hours—the tongue becoming more moist, and covered with a white fur—distension of the abdomen ceases to progress, and after a time diminishes. The use of the oil should be continued under these circumstances; but the dose should be gradually diminished.

Tonics and *stimulants* may be absolutely essential on account of debility attending the advanced stage of the disease, generally about the third week. When the pulse is slow and feeble, the skin cool, the tongue and teeth encrusted with dark sordes, at an advanced period of the fever, then stimulants are obviously necessary. But even when the pulse is feeble, but yet frequent, and the skin hot, stimulants are even then known to be of service; but it is necessary to administer them with great caution, and to watch the effects constantly and closely. If their use is found to augment the heat of the skin, and to increase the frequency of the pulse, and to aggravate the delirium or stupor, it is then necessary to suspend their use. They are

known to be doing good service, however, if they lessen the frequency of the pulse, and increase its fulness and strength; if the skin becomes cool and moist, and if the delirium is subdued or moderated; and especially if refreshing sleep be procured. Dr. Wood recommends the use of wine whey, prepared by adding *one quart of good sherry wine to two quarts of boiling milk*, and straining after coagulation.

Of this a table-spoonful or more may be given every hour or every two hours. If the strength is greatly reduced, it may be necessary to give pure wine or brandy; or even sulphuric or chloric ether in cases of great prostration. Opium is also a useful stimulant. It may be given when the pulse is not full nor strong, and when cerebral symptoms do not exist. In the later stages it may be given in doses of half a grain, or a grain, every four, six, or eight hours. It is known to be acting beneficially when it promotes sleep, subdues nervous excitement, and induces gentle perspiration. Carbonate of ammonia is objectionable, as it may irritate the bowels and increase the diarrhoea.

The diet is of the utmost importance to be attended to in cases of typhoid fever from the very commencement of the disease. It ought then to be both food and drink combined, in the form of a light nutritious liquid. Barley-water, rice-water, toast and water, thickened more or less with solutions of tapioca, sago, arrow-root, *the juice* of sweet fruits, or the very soft pulp of fruits, or the pure jelly of ripe fruits; but fruit in its crude state is to be strictly withheld. It is necessary, as a rule, to give food at certain intervals and in certain quantities. A wine-glassful should be given at least every two or three hours, according to the state of digestion and the demands upon the strength of the patient. It may be that the patient is unable to swallow, from the dry and shrivelled state of his tongue. Before offering him food or drink, therefore, the nurse should put a tea-spoonful of lemon juice and water into his mouth. She must then wait a minute or so, until the fur upon the tongue and mouth is softened and moist, after which the patient will often drink or take his food with ease. Milk in small quantities frequently repeated will be found an excellent diet; and animal broths and jellies may ultimately be given. The *extractum carnis*, as prepared by Liebig, is a most valuable nutriment for typhoid fever patients.

The utmost caution is necessary as to diet and aperients during

convalescence; *first*, as to opening the bowels, castor oil or simple enemata are the only means which should be resorted to; *secondly*, as to diet, no flesh meat should be allowed till at least seven days after all the febrile phenomena have passed away, and the food should be as free as possible of excrementitious matter. Malt liquors should not be taken *before* food.

TYPHUS* FEVER.

LATIN, *Febris typhus*; FRENCH, *Typhus*; GERMAN, *Exanthematischer Typhus*—Syn., *Fleckfieber*; ITALIAN, *Tifo*.

Dermo
Definition.—A continued fever, attended with sluggishness of intellect and confusion of thought, followed and accompanied by an eruption on the skin, of a rubeoloid character, appearing generally from the fifth to the eighth day, at first slightly elevated, and disappearing on pressure, but after the second day persistent, and remaining persistent for eleven or twelve days. Languor and weariness, prominent from the first, gradually pass into stupidity, oblivion, and complete prostration; which, in still more extreme cases, pass into somnolence, stupor, and sometimes coma, when prostration becomes profound. The disease may terminate favourably from the thirteenth to the seventeenth day, the average duration of the attack being about twenty-one days. If the disease proves fatal, it is generally between the twelfth and the twentieth day, leaving no specific lesion beyond hypercæmia, softening of the heart and contractile fibre-structures, and atrophy of the brain.

* As the term typhus is very variously used, and sometimes vaguely, it is necessary to state precisely the meaning of the word. This cannot be more clearly, distinctly, and concisely expressed than in the words of Dr. Wood.

The disease now defined is sometimes called *typhus*, and sometimes *typhus fever*. In the first instance the term is used “substantively, in the latter adjectively, just as we say ship fever, jail fever, &c. But a state of system, identical or closely analogous with that which characterizes *typhus* fever, is frequently met with in other febrile diseases, as a mere incidental accompaniment. To this morbid state the epithet *typhous* or *typhoid* is applied, the latter being preferred to the former when it is wished to imply resemblance only, and not sameness or identity. Thus we speak of a *typhous* or a *typhoid* condition of remittent fever, yellow fever, small-pox, measles, pneumonia, dysentery, or, with greater brevity, *typhous pneumonia*, *typhous dysentery*. This latter phraseology, however, generally implies a more thorough incorporation of the *typhoid* element with the principal affection than *typhoid pneumonia* or *typhoid dysentery*, which merely implies a resemblance to the *typhous* state occurring in these diseases.”

Historical Notice.—The first authentic accounts of typhus fever are to be found in the early British chronicles. It is described as having spread in our courts of justice, giving rise to what was termed “the black assizes.” The last black assizes happened at the sessions of the Old Bailey in 1756, when the lord mayor, two of the judges, and several eminent persons died, infected by the prisoners. This fever has had many popular appellations, having been known as the *jail fever*, *hospital fever*, *ship fever*, *putrid fever*, *brain fever*, *bilious fever*, *spotted fever*, *petechial fever*, *camp fever*. We are indebted, however, to Pringle and to Fordyce for having shown that these supposed different fevers are identically the same, and have no such essential differences as to constitute them distinct genera.

Typhus fever is the grand scourge of armies in temperate climates, just as cholera and yellow fever have been the destructive agents in the tropical wars, (Parkes “On the Causes of Sickness in English Wars,” *Journal of Royal United Service Institution*, vol. vi.) Wherever men are closely crowded together in ill-ventilated unwholesome dwellings, typhus is sure to appear. It has often passed from the army to the civil population, and has thus dispeopled towns, and even great districts of country. But its ravages in the English army have never been comparable to those which have occurred in foreign forces, as the statements of Murchison and Parkes fully demonstrate:—“In the year 1489 no fewer than 17,000 of the troops of Ferdinand, then besieging Granada, were destroyed by a spotted fever, to which the Spaniards applied the same name that they afterwards gave to typhus. In 1552 a petechial fever devastated the army of the Emperor Charles V. during the siege of Metz. In 1556 the notorious ‘*Morbus Hungaricus*’ appeared in Hungary in the army of Maximilian II., and thence spread over the whole of Europe” (MURCHISON, l. c., p. 21). “In 1620 the Bavarian army in a few months lost in Bohemia not less than 20,000 men from spotted typhus; and the disease, being carried into other parts of Germany, obtained the name of ‘the Bohemian disease.’ In 1628 and 1632 the Swedish army under Gustavus Adolphus carried typhus into Northern Germany, and the population was so destroyed that, fifty or sixty years later, villages were left without inhabitants, (PARKES, l. c.) In the spring of 1643, while the Earl of Essex was besieging the town of Reading, this disease broke out in

the army of the Parliamentary General, and in the garrison commanded by Charles I.: it was communicated to the inhabitants of the surrounding country, and proved very fatal, (MURCHISON, l. c.) The wars of Louis XIV. were always followed by this disease, and the losses of the French army were enormous (PARKES). In 1799-1800 an epidemic of typhus occurred at Genoa, when the garrison was besieged by the French, and half famished; and the French army, during their retreat from Italy, communicated fever to the inhabitants of fifteen towns and villages where they halted on the route (FODERE). It was during the first fifteen years of the present century that the greatest ravages of typhus have been recorded, especially in the armies of Napoleon, and among the population of the countries which were the seat of war. It always became developed under circumstances of misery and privation, and was particularly prevalent and fatal among the inhabitants of besieged cities—as, for example, Saragossa and Torgau, Dantzic and Wilna, in 1803—and which told with such awful severity upon the famished French troops during the retreat from Moscow in 1812 and 1813 (MURCHISON). When Sir John Moore's army landed from Corunna, typhus became epidemic in the military hospitals in the south of England (Cheyne, *Dub. Hosp. Report*, vol. ii., p. 3). In May, 1812, the Bavarian army serving among the French numbered 28,000 men; in February, 1813, there were only 2,250 men under arms. The great destroyer was typhus. In August, 1813, the first Prussian army consisted of 37,728 fighting men, having lost 16,000 men by the sword, and 10,000 men by disease, almost entirely typhus. In Mayence alone, of 60,000 French troops composing the garrison in 1813-14, there died of typhus in six months 25,000 men (MURCHISON, p. 224). The last great ravages of typhus in armies which attracted public attention were those which occurred in the French and Russian armies in the Crimea during and after the Capture of Sebastopol. Typhus had prevailed in the winter of 1854-55 amongst both the English and French troops; but in the following winter it was mainly confined to the French and Russian armies. In the spring of 1856 it was computed that more than 17,000 men of the French forces perished in less than three months; and the highest authority stated that the safety of the whole French army was endangered by the outbreak (PARKES and MURCHISON).

According to Dr. Parkes, typhus fever occupies the fourth place among the causes which have produced disease in the British army.*

Phenomena and Symptoms.—Typhus fever attacks persons of both sexes and of all ages, from early infancy to extreme old age, and its advent is somewhat sudden.

After a longer or shorter duration (generally a few days) of unpleasant sensations—in which general soreness, uneasiness, and fatigue without cause, loss of appetite, and disturbed sleep, are the prominent phenomena—the disease begins and advances gradually. It is not possible in all instances to fix the precise time of the commencement of the attack; but in the majority of cases the patient is seized with chilliness, which sometimes amounts to a rigor, usually followed by heat of skin, and occasionally by sweating, pains in the back and limbs, and frontal headache. This headache is a constant symptom, which ceases usually about the tenth day, and always before the fourteenth. During two or three days the chilliness and rigors occur at irregular intervals. The patient alternately hovers over the fire or desires to move from it; and although the skin at the time may be felt hot and burning, he still lingers near the fire-place, and yet again soon complains of the heat of the room; so that he feels when near the fire hot and oppressed, and when away from it chilly and uncomfortable. Loss of appetite, and more or less thirst, exist from the

* These causes Dr. Parkes arranges as follows:—

1. A defective commissariat, especially as to food and fresh vegetables, causing diseases, but mainly predisposing to many more—*e. g.*, malignant malarious fevers, scurvy, and bloody flux. Carthagera, 1741; Burmah, 1824; China, 1840.

2. Undertaking military operations in an unhealthy site, and with an unhealthy season impending. Carthagera, 1741; San Domingo, 1796; Walcheren, 1747; Java, 1811; American War, 1814; Bulgaria, 1853-54.

3. Exposure to cold, with insufficient clothing and food, giving rise to catarrhs, slight dysentery, rheumatism, and inflammations. Wars of 1742-1760; Crimea, 1854.

4. Propagation of typhus poison, favoured by bad ventilation, overcrowding, and filth. Examples as above detailed.

5. Similar propagation of putrid dysentery. Indian campaigns.

6. Propagation of typhoid fever poison and cholera, through the bad sanitary condition of camps, and the occupation of old camping grounds. Egypt, 1801; Bulgaria, 1853; India.

7. The enlistment of boys as soldiers, whose bones are not yet matured—in place of full-grown men at least twenty-one years of age. Crimea, 1854. [See a short publication *On the Growth of the Recruit and Young Soldier*, by William Aitken, M.D.]

8. Want of cleanliness, excessive use of spirits, and debauchery.

first; the tongue is white, large, and pale, but is afterwards covered with a yellow-brown fur, and is sometimes tremulous, indicating the early loss of muscular power and control. The bowels may be confined or regular; the urine is scanty and high-coloured; and nausea with vomiting are often among the earliest symptoms. If sleep is obtained, it is disturbed by dreams, or by the occurrence every few minutes of sudden starts. It is consequently unrefreshing; and although the patient may have appeared to sleep for hours, yet he feels that he has not slept, and declares that he has never closed his eyes. This is the *coma-vigil* of Chomel. On the other hand, there is sometimes a constant tendency to heaviness and drowsiness. The attention cannot be fixed, and the mind ceases to think. A peculiar symptom may now become expressed, to which Dr. Jenner has given more appropriately the name of *coma-vigil* than to that symptom which Chomel has so named. In the *coma-vigil* of Jenner "the patient lies with his eyes open, evidently awake, but indifferent or insensible to all going on around him." This symptom occurred in one-fifth of the fatal cases observed by him. Bodily weakness becomes extreme, and the patient takes to bed by the second or third, and not unfrequently on the first day. While there is absolute loss of muscular power and control, there is at the same time an amount of great exhaustion, disproportionate by its severity to the muscular action. Giddiness and noise in the ears are amongst the earliest and most loudly complained of symptoms. The debility increases rapidly, so that by the seventh day the patient can rarely leave his bed without some assistance. By this time also the want of control over the muscular movements becomes more decided; the legs and arms shake when raised, and the tongue trembles when protruded. The impairment of the mental powers manifests itself in a variety of singular ways. Memory becomes deficient,—the ideas of time are such that it is always supposed to be prolonged. If an event is impressed upon the patient's mind, he will remember it, and it alone. This mazy state of the intellect soon passes into delirium, which becomes manifest first between waking and sleeping, then by night, and finally by day and night. When delirium first sets in, the patient is able to correct himself; if he is made to think, he becomes conscious of his mental error; but this power is soon lost, and delirium becomes predominant.

About the tenth day of the disease, sometimes earlier, the headache ceases simultaneously with the commencement of the delirium; and if it should continue with the delirium, it suggests the probability of some commencing secondary lesion within the cranium, to which special attention must be immediately directed.

The Eruption of Typhus Fever.—About the fifth to the seventh day of the disease the characteristic eruption appears on the skin. It consists of,—(1.) Distinct spots; (2.) A subcuticular rash.

(1.) The *maculæ*, *mulberry*, or *rubeoloid rash*. On the first appearance of this eruption it consists of very slightly elevated spots of a dusky pinkish-red colour, somewhat like the stains of mulberry juice. Each spot is flattened on the surface, irregular in outline, with no well-defined margin, and fading insensibly into the hue of the surrounding skin. The spots *disappear* completely on pressure, resuming their distinctive appearances as the pressure of the finger is withdrawn; and they vary in size from a point to three or four lines in diameter. The largest spots appear to be formed by the coalescence of two or more smaller ones; and the shape of the larger spots is more irregular than the smaller ones. After one, two, or three days these spots undergo a marked change. They no longer remain elevated above the surrounding cuticle. Their hue becomes darker and more dingy than at their first appearance. Their margins become more defined, especially on the posterior surface of the body; and when the finger is firmly pressed on them, they grow paler, but do not entirely disappear. Thus they are said “to fade under pressure;” but they cannot be entirely obliterated, a stain of the cuticle remaining to indicate where they are. A still further change may take place in severe cases. The centres of the spots may become dark purple, unaltered in appearance by the firmest pressure, although their circumferences may fade; or the entire spot may change into a true petechia, becoming of a dusky crimson or purple colour, quite unaffected by pressure, with a well-defined margin, and level with the surface. The spots of such an eruption are generally very numerous, close together, and occasionally almost covering the skin. Sometimes, however, they are very few in number, and situated at some distance from each other; and not to be distinguished at first from the *rose spot* eruption. The *mulberry eruption* usually occupies the trunk and extremities, but is occasionally limited to the trunk, and may now and then

on trunk
back of
hands -
Jenner
almost
never on face
1/2 inch up

be observed to extend to the face. After the first, second, or third day after the eruption is apparent, no fresh spots appear, and each spot remains visible from its first eruption till the whole rash vanishes—that is, till the termination of the disease. When very numerous, the eruption, viewed as a whole, has not an equal depth of colour. Some places are much paler than others, and the spots have a dull appearance, as if seen through the cuticle. A mottled aspect is thus sometimes given to the skin, on which the darker spots are seated; and hence (2.) A *subcuticular rash* has been also described, which is deepest coloured on the most depending parts of the body. From this circumstance the eruption sometimes resembles *measles* so closely as to be distinguished with difficulty from the eruption in that disease. When the spots on the back are of a much deeper hue than those on the anterior surface of the trunk, the skin is at the same time so much congested at the back that slight pressure with the finger leaves a white mark, which slowly returns to its dusky red colour. The eruption of the mulberry rash usually appears from the fifth to the eighth day of this disease, and subsides between the fourteenth and twenty-first days (DR. JENNER).

Age seems to exert a considerable influence over the eruption; and the following rule has been laid down in relation to this modifying circumstance:—In 100 typhus patients under fifteen years of age the rash will be absent in 25. In 100 typhus patients between fifteen and twenty-two years of age the rash will be absent in 14. In 100 typhus patients above twenty-two years of age the rash will be always present.

The spots of typhus fever continue ineffaceably persistent after death.

Tongue At the termination of the first, or commencement of the second week, the tongue has a large and swollen appearance, grows dry in the centre, and at the same time its white fur is replaced by pale dirty-brown mucus.

About the ninth or tenth day, or even earlier, the delirium becomes decided, sometimes violent, and always unquiet, although the attention may still be fixed by a sharp question. At this time the patient is in some cases violent, and, unless restrained, leaves his bed to wander about the room. His expression gradually comes to resemble that of a man unwilling to be roused from half-drunken slumbers. It now betokens complete stupidity,

oppression, and decided prostration. The complexion, dull and dirty from the first, in the course of the second week becomes absolutely muddy, the conjunctival membranes injected, and the pupils contracted; and the danger of febrile coma, which may supervene, seems very much in proportion to the contraction of the pupil (W. T. GAIRDNER). The face is now often flushed—the flush being dingy and pretty uniform over the whole countenance; but occasionally somewhat more marked on the cheeks than elsewhere.

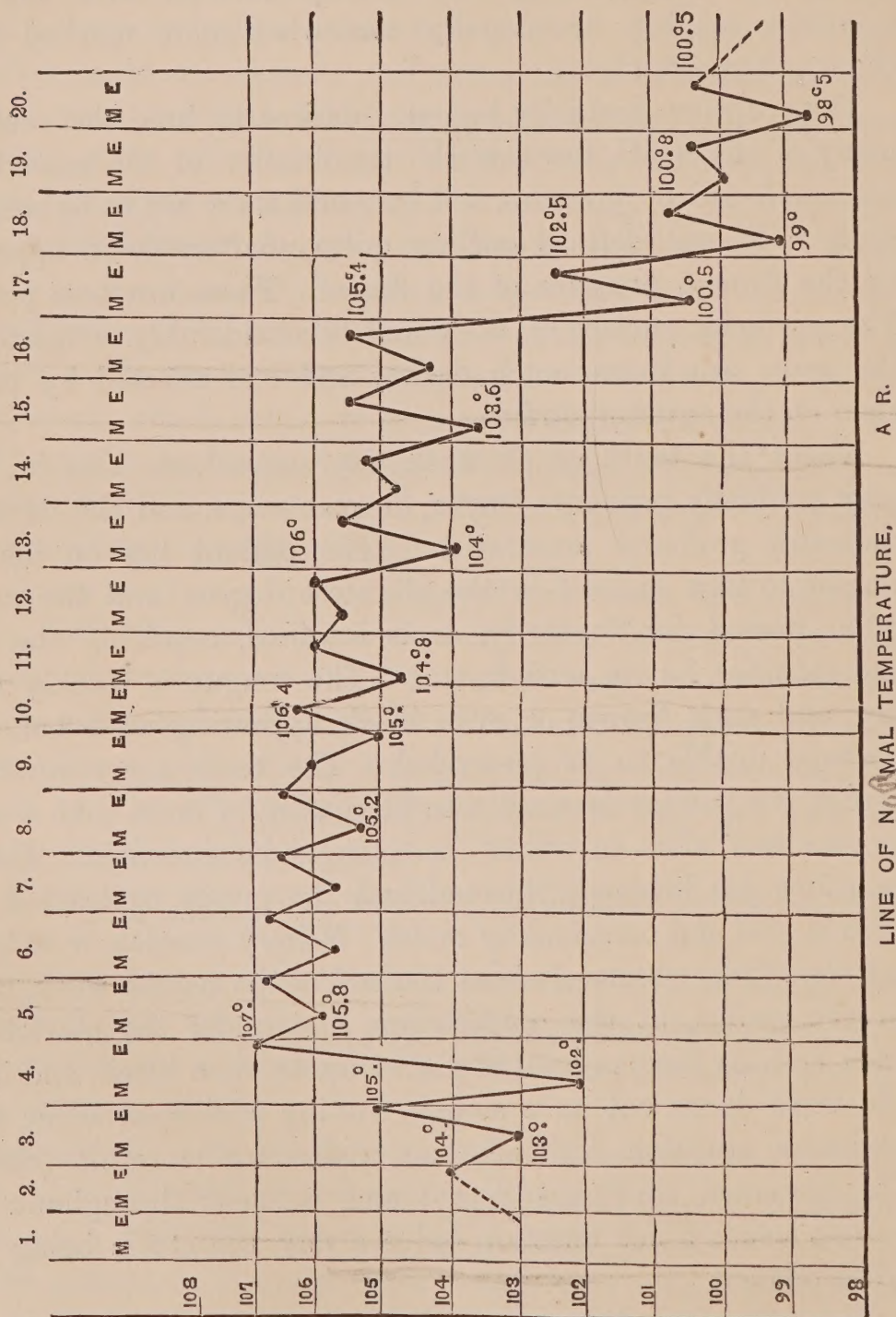
The eruption gradually becomes darker in hue, the centres of many of the spots, towards the termination of the second week, are unaffected by pressure, and here and there are to be seen some spots with well-defined outline, quite unalterable in appearance by the firmest pressure of the finger. These are true petechiæ. The posterior surface of the trunk is considerably congested, and the spots are there much darker and less affected by pressure than on the anterior surface.

About the tenth or eleventh day somnolence sets in, which may gradually pass into stupor, or even coma, and the expression indicates profound prostration. The patient lies on his back, unable to turn himself in the slightest degree, and the urine is often passed involuntarily, or is retained, requiring the use of the catheter for its withdrawal. The tongue is thickly coated, dry, and dark brown, or even black, appearing as if baked, and perhaps unable to be protruded. The teeth are covered with sordes, the patient is unable to be roused for more than a minute or two, and when so roused he mutters incoherently. The conjunctivæ are intensely injected, and the pupils contracted. The skin is cool and occasionally moist. Miliary vesicles, or sudamina, are sometimes observed about the end of the second week, usually in the groins, at the epigastrium, and under the clavicles. If such vesicles become hard at the summits, then black, and if then the mass drops out as a slough, leaving a circular ulcer, such a vesicular eruption forebodes an unfavourable result (STOKES). The abdomen continues flaccid and indolent throughout. The bowels usually act once or twice a day, the stools being somewhat relaxed.

The pulse, from the outset of the disease, is quickened, and it increases in rapidity in cases which terminate fatally, ranging from 100 to even 150 in a minute; or, after reaching a certain

point, its frequency as gradually subsides till health is restored. Cases in which the pulse is remarkably slow are usually cases in which the prostration becomes extreme. In the milder and uncomplicated cases the maximum rate of the pulse is reached before the eighth day, and continues for two or three days, at least, at the maximum rate—commencing to decline gradually

RANGE OF TEMPERATURE IN A SEVERE CASE OF TYPHUS FEVER. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).



about the tenth, eleventh, or twelfth day. In the more severe cases the pulse keeps up very high till the thirteenth or fourteenth day, when, if the disease is about to end favourably, there is a

A sudden and marked fall (say from 120 to 96, or from 100 to 84), indicating a decided crisis (PERRY).

The skin throughout the whole course of typhus fever is often particularly sensitive, the slightest touch occasioning pain. The heat of the skin conveys also a burning pungent sensation—the temperature ranging from 102° to 107° Fahr. Greisinger, of Zurich, after insisting on the non-identity of typhus and typhoid fevers, has described the ranges of temperature in cases of typhus as particularly diagnostic and characteristic (*Arch. der Heilk.*, vol. ii., p. 557, 1861). Dr. Cheyne, of Dublin, recorded 109° Fahr. as the highest he observed, and a few days before death he observed the temperature to fall to 95° Fahr.

The course of typhus fever, although it may have some features in common with that of enteric or typhoid, yet shows great and numerous differences. In typhus, the fever, as denoted by the temperature, is maintained continuously for a longer time without interruptions.

If the temperature at the commencement before the fourth day does not exceed on any evening 103·5° Fahr., the fever may be expected to run a mild course; and more especially if the increase of temperature takes place moderately, and is of limited daily duration during the beginning of the second week. In mild cases a small abatement of temperature takes place on the fourth day, which becomes more obvious by the seventh day; and this abatement is followed, at the commencement of the second week, by a small re-increase of very limited duration, after which the tendency to further decrease re-appears, and by the end of the second week, or the beginning of the third, the normal temperature is again attained. On the other hand, in severe cases the fever continues with great intensity, at least to the twelfth day, and mostly throughout the whole of the second week.

Defervescence rarely takes place before the first half of the third week. In very severe cases the temperature may rise above 104·7° Fahr., and it frequently reaches 106° Fahr. or more. The maximum of temperature is usually attained before the ninth day (GRIMSHAW), or on the ninth day (WARTER); and according to Grimshaw the extreme height of the thermometer in typhus seldom exceeds 104°, occasionally reaches 104·5°, and may, in exceptional cases, rise to 105°—a conclusion nearly coinciding with the observations of Dr. Perry, of Glasgow. The differences between the morning and the evening temperatures amount,

Range between morning & evening temperatures
1 - 1.5 Fahr

MB during the fastigium (from the middle of the first to the middle of the second week), rarely to more than 1° Fahr.; and from the middle of the second week to a difference of about 1.5° Fahr. Greater differences happen only temporarily—a character which distinguishes typhus from enteric or typhoid fever, so far as ranges of temperature are concerned. But the difference between typhus fever and enteric or typhoid fever is rendered still more striking by their respective modes of defervescence. In typhus fever the defervescence shows no gradual remissions, as in enteric or typhoid fever; on the contrary, the defervescence of typhus fever is invariably sudden. When the fall is great (3° to 5°) as well as sudden, critical diarrhoea may be severe and serious, as in some of the cases detailed by Dr. Grimshaw; and a sudden fall of temperature to a range below the normal, other symptoms being serious, is a bad sign, and may forebode death. It takes place precipitantly, and the normal temperature is reached sometimes in one night, mostly in twenty-four or thirty-six hours.

MB The disease generally terminates, if it proves fatal, from the twelfth to the twentieth day; and before death the prostration increases to the last degree. The average duration of Jenner's fatal cases was fourteen days; of Reid's, thirteen days. *Subsultus tendinum*, or involuntary twitchings of the muscles of the face and arms, make their appearance. The face becomes dusky or even livid, and the breathing very quick, the pulse becoming so rapid and feeble that it can scarcely be felt. Some want of resonance of the most depending part of the chest may often be observed at this stage of the disease. The respiratory murmur at the same part becomes muffled, as if heard through a covering, and there is sometimes a little coarse, unequal crepitation. The urine, which is now secreted in large quantities—from three to four pints daily—is restrained, or passed into bed with the stools involuntarily. The skin is bathed in a profuse sweat, and the temperature is apt to fall below the natural standard. The patient lies on his back unable to move, or he sinks towards the bottom of the bed if his head be in the least elevated. Towards the middle or end of the second week a slough may form on the lower end of the spinal region, or on the region over the posterior spine of the ilium.

Bedrney For a day or two before the fatal termination, the condition termed *coma-vigil* may come on. In this condition the patient never sleeps. He lies on his back with his eyelids widely

separated, his eyes staring and fixed in vacuity, his mouth partially open, his face pale and expressionless. He is totally incapable of being roused to give a sign of consciousness, the breathing is often scarcely perceptible, the pulse rapid and feeble, or unable to be felt, the skin cool, perhaps bathed in perspiration. Life is only known to have ceased by the eye losing its little lustre, and the chest ceasing to effect its slow and feeble movements. Dr. Jenner has never seen recovery from this condition. Death generally takes place without any return to consciousness, and by syncope rather than coma (MURCHISON).

If the disease should terminate in recovery, the improvement in the condition of the patient is frequently sudden. Some time between the thirteenth and the seventeenth day he may fall into a profound quiet sleep, lasting for several hours; and generally after from twelve to twenty-four, or even more hours, he awakes decidedly improved in all respects—indeed, quite another man. At first he is bewildered or confused, and wonders where he is; but he may recognize his attendants and friends, and is conscious for the first time of his extreme debility. The complexion is clearer, the delirium has disappeared, the pulse has fallen in frequency and gained in strength, the conjunctivæ are no longer injected, the tongue is moist at the edges; there is perhaps a little appetite, the skin is softer and moist, and the spots paler. His limbs retain their sensibility, but when he attempts to move them, they seem at first as if separated from the body, so great is the prostration induced by typhus fever (MURCHISON). In a few days the tongue cleans completely, the appetite becomes ravenous and insatiable, and the patient rapidly regains strength. Dr. Jenner considers the duration of the disease to be measured by the duration of the eruption; and the average duration of cases that recover, he states to be from fourteen to twenty-one days; although, not unfrequently, in very mild cases, the fever terminates before the fourteenth day. After twenty-one days local lesions sufficient to cause death were always discovered in fatal cases of *typhus*. In other words, after the twenty-first day, death does not then occur from the fever alone, as may be the case before the twenty-first day. There are two very opposite circumstances under the influence of which the date of the first appearance of the eruption is changed, and its duration shortened. These are,—(1.) A very mild attack of the specific disease; (2.) The development of severe local complications in the course of

the specific disease. There are cases of *typhus fever* which appear to die within a limited period after the outset of the illness, from the direct action of the poison on the blood or nervous system, and with the entire absence of local lesion, so that even the skin is not affected with eruption.

Condition of the Blood.—In *typhus fever* the microscopical characters of the blood are often such as to prove a marked deviation from its normal state. Amorphous heaps of red discs replace the normal rouleaus, and the adhesion of the red discs to each other, in the imperfectly formed rouleaus, is far less complete and long continued than in healthy blood. The red discs part with their colouring matter more easily and dissolve more rapidly than they do in their normal state. This is shown by the red serosity found in almost every serous cavity, the deep dusky-red hue of the flesh, and of every structure in contact with the blood. The blood drawn during life, or found after death in the vessels, is loosely coagulated or absolutely fluid (DR. JENNER). It is also more apt to become putrid, when taken from the body during life, than healthy blood, or than blood in other diseases. According to Lehmann, the salts are increased rather than diminished; and there is good reason for believing that the unnaturally fluid state in typhus fever results from an abnormal amount of ammonia, possibly derived, as Dr. Murchison suggests, from the decomposition of urea; and there is evidence, as Drs. Richardson and Murchison have shown, that the blood of typhus fever contains an increased amount of ammonia.

Secondary Lesions and Complications of Typhus Fever.

1. **Convulsions and Cerebral Affections.**—The most formidable, and fortunately the most rare, is the occurrence of convulsions. When they do occur, the case almost invariably proves fatal, unless the convulsions occur in a patient who has suffered from epilepsy; and the subjects of them seldom present any morbid appearances after death sufficient to account for their occurrence. In *The Edinburgh Monthly Journal* for June, 1848, the details of six cases of typhus fever are given, five of which proved fatal by convulsions in different wards of the general hospital there, and all of them within a period of twenty-one days, in the months of January and February. The following are the days of the fever at which convulsions are stated to have occurred, the number of hours they continued to recur, and the apparent mode of death:—

No. of Case.	Day of Fever.	Number of Hours the Convulsion Recurred.			Mode of Death.
1st. <i>Edin. Mon. Journal,</i>	7th		2		Coma.
2d. ,, ,,	11th		5		Coma.
3d. ,, ,,	15th		6		Coma.
4th. ,, ,,	14th		24		Coma.
11th. Of Mary G—, re- lated by Dr. Jenner,	11th to 13th		72		Coma.
13th. Of Thomas B—, re- lated by Dr. Jenner,	9th		36		Coma.

The occurrence of convulsions in such cases may be fairly referable, in the present state of our knowledge, to the morbid condition of the blood in *typhus* fever, and the altered condition of the nervous system which ensues; and probably they have always a uræmic origin. With reference to the absence of any appreciable lesion in the brain, as in these cases, it may be remarked that our usual instruments of research, applied to the nervous texture, are insufficient in all instances to indicate disease, even where it does undoubtedly exist. There are, for instance, physical conditions of texture, which are of the utmost importance in pathology, such as the specific gravity, and which are appreciable by the proper means and instruments of research, even when the tissue of the organ presents to our senses of sight and touch no external evidence of disease.

The cerebral complications are generally attended with what are commonly called "head symptoms," and are preceded by long-continued high temperatures. Dr. Jenner very emphatically calls attention to the fact, that the continuance of the headache complained of spontaneously after the commencement of delirium is generally indicative of increased vascular action within the cranium. It may also be noticed that the headache which precedes the delirium is often in such cases of a very severe and constant kind, the face being sometimes pale and sometimes red, and greatly expressive of the distress the patient suffers. The eye, haggard or brilliant, with its conjunctiva injected and its pupil contracted, is painfully sensible to the light, and is therefore generally closed. The least noise is insupportable, and the patient is troubled with noise in his ears. His temper is altered, and his answers short and fretful. This condition is that of increased excitement, but not as yet of delirium, and, supposing the membranes of the brain to be inflamed, denotes diffuse inflammation of those tissues. At the end of a period of time, varying from two to ten days, the patient becomes delirious. His delirium may

- (1) Convulsions & Cerebral Symptoms
(2) Hypostatic Pnea
(3) Sanguine of Lung
(4) Softening of Heart

assume every character,—joyous or melancholy, furious or tranquil; and in some cases he wanders from subject to subject, while in others he incessantly recurs to the same theme, and even to the same few words. In others, though the cases are few, the disease assumes every character of insanity; and, if permitted, the patient, confined in a strait waistcoat, presents the extraordinary spectacle of being able, in typhus fever, to walk about the wards. The phenomena of this stage show that the inflammation of the membranes of the brain has extended to the substance of the brain itself. The commencement of effusion is indicated by the active delirium changing into a low muttering (*typhomania*), by the patient no longer requiring restraint, by his muscles becoming spasmodically affected with slight twitchings, or *subsultus tendinum*, showing how rapidly the nervous power is exhausted, and how feebly supplied; also by the pupil of the eye becoming expanded or contracted; by the fæces being passed involuntarily; by the urine being retained; and by the rapid grouping of those other symptoms so happily described by Shakspeare as “the stony coldness of the feet creeping upward and upward,” “the babble of green fields,” and the “fumbling of the bed-clothes,” all indicative of approaching death. Intra-cranial serosity is generally decidedly increased; and hæmorrhage into the arachnoid occurred in one-eighth of Jenner’s fatal cases. When the patient recovers, however, from this stage of cerebral complications, the appetite improves, the pulse becomes fuller and steadier, the countenance more tranquil, the mind firmer, and the sleep natural, till at last convalescence is fully established.

The respiratory movements are often influenced by this cerebral condition. In the first week they do not exceed twenty or twenty-four in the minute; but when delirium supervenes and the pulse increases in frequency, they often rise to thirty or more, without any pulmonary lesion. In cases of great cerebral disturbance the respirations become sighing, irregular, spasmodic, or jerking, and then coma is apt to supervene. The “nervous respiration” of Dr. Corrigan, or what is sometimes also called “cerebral respiration,” is denoted by a blowing or hissing sound in breathing, the lips being kept closed; the cheeks are distended, the nostrils dilate with each expiration, and the breath is forced through the closed lips with a puffing, blowing noise. Such breathing is irregular, a long pause being followed by a deep inspiration, and subsequently by short respirations in rapid suc-

cession. In some cases the action is entirely diaphragmatic, the respiratory muscles of the trunk being paralyzed (MURCHISON). The air expired has a disagreeable odour, most marked in the advanced stage of severe cases. It resembles the *typhus odour* exhaled by the skin, and which has been compared to the "odour of rotten straw," to the "smell of mice, deer, and certain reptiles," or to the smell of "the leaves of rue when rubbed between the fingers." By some it is spoken of as "pungent, ammoniacal, and offensive;" but is not to be confounded with the smell from urine passed in bed. It seems to be, however, a smell *sui generis*, as Murchison very justly observes; and nurses experienced in typhus fever are quite familiar with it, and are able to distinguish cases of typhus fever by this peculiar typhus odour alone, which is always strongest in damp weather, and when the ventilation is bad; and it is highly probable that the typhus poison is contained in this odoriferous substance. The expired air of typhus patients contains a smaller quantity of carbonic acid and a larger amount of ammonia (MURCHISON, pp. 134-137).

2. **Secondary Pulmonic Complications** are not uncommon in *typhus fever*, in the form of *pneumonia*. The congestion of the blood in the posterior parts of the lungs may give a tendency to this; and its presence may be suspected from the livid expression of the face, the existence of cough with rusty expectoration, the diagnosis being confirmed by the usual auscultatory means. It is seldom that pain is complained of. The part of the lungs affected for the most part is that which rests against the hollow of the fourth, fifth, and sixth ribs, between their tubercles and angles; the position of the patient appearing to determine the place of consolidation. Such consolidations are not to be regarded as analogous to what we see in an ordinary pneumonia, occurring in an otherwise healthy person. The incubation of the lesion is latent, and the symptoms are masked, and the nature of the complication is only to be recognized by careful physical examination and determination of temperature, which suddenly rises and continues high. The full expression of the morbid state is often for some time undecided; the exudation, being of a serous nature, is slow to solidify on the one hand, and yet the symptoms of resolution do not appear on the other. Dr. Hudson, of Dublin, attaches some importance to a certain tympanitic resonance, which becomes manifest over the diseased lung, as a sign of the existence of the pulmonic lesion. He describes it as "a tympanitic clearness

over the solidified lung, without air being present in the pleura." Dr. Lyons explains this abnormal clearness as the result of the increased pressure of the respiratory column of air in the permeable portions of the pulmonary lobules, which become expanded beyond their natural volume, and thus a condition of temporary emphysema is produced, which yields a clear sound on percussion (Dr. Stokes, in *Medical Times and Gazette*, May 26, 1855).

In some cases of pulmonary lesions there appears to be a combination of circumstances which leads to a fluid or purulent state of the diseased part, resembling the third stage of pneumonia as described by Laennec. The conditions which lead to this form may be stated to be,—(1.) A sudden exudation and abundance of fluid matter; (2.) A great amount of tissue involved; (3.) Diminished vascularity and consequent (4.) Abeyance of absorption, tending to (5.) Increased fluidity of the diseased part; (6.) Breaking up or solution of the young and growing elements. A lung in this condition seems to have passed, as it were, at once into this state, without any well-marked hepatization.

3. **Gangrene of the Pulmonary Tissue** is by far the most formidable of the thoracic secondary lesions of *typhus fever*. The hepatization of the lung is not, as in the last instance, obscure, but the consolidation is at once sudden, complete, and extensive, involving perhaps the greater part of a lung, and coming on without any marked physical signs different from what are to be heard in the simple congestion of *typhus*. A gangrenous cavity forms in the substance of the solidified mass, and is only indicated by the fetid expectoration and the accompanying physical signs of a cavity. Large eschars are apt to form towards the pleural surface, surrounded with well-defined lines of demarcation where separation of the slough proceeds. In this gangrenous slough every simple element of the pulmonary tissue becomes disintegrated, almost perfectly liquescent; and sometimes it happens that the gangrenous cavity does not communicate with the bronchial tubes, and then the morbid state is difficult to diagnose, and its existence is often unknown till after death. With physical signs, the expression of the countenance of the patient is often highly suggestive. It suddenly becomes small, pinched, contracted, ghastly, miserable, and death-like. The eyes are sunk and void of lustre; and, along with languor, the patient feels nausea, and sometimes vomits. There may be several distinct gangrenous centres, as if the lesion had been, from the first, disseminated or lobular.

4. Secondary Cardiac Lesion.—This lesion assumes the form which Dr. Stokes has called “typhus softening of the heart.” He is inclined to consider that the muscles of the larynx and the circular muscles of the trachea are sometimes similarly affected, as well as the involuntary muscles generally. This complication has for the most part occurred when there was a great amount of the secondary bronchial disease. The wasting of the involuntary muscles is always great in typhus. In the heart it is more obvious than in the arterial or systemic portion. WB

The cardiac phenomena of typhus (adynamic) are chiefly indicated by a diminution of the impulse, and an impairment or loss of the first sound—the impulse diminishing progressively from the fifth or sixth day to the termination of the disease—while the systolic sound becomes daily more feeble or quite inaudible, leaving the second sound clear and distinct.

The poison, however, does not necessarily make itself manifest through all the series of local secondary affections already referred to. Thus, in one year the lungs will be attacked in every case; in others, the bronchial membranes or the membranes of the brain; while in other years such attacks will be rare,—the exception, and not the rule of the disease.

Prognosis.—The occurrence of certain phenomena, or the presence of certain symptoms, indicating the existence of complications, are the main guides to an opinion. These may be arranged in the following summary under three heads, namely:—

1. Combinations of Symptoms and Phenomena which are of extremely unfavourable import.—(1.) A presentiment of death on the part of the patient; (2.) A pulse of 120, which at the same time is soft and compressible, small, fluttering, irregular, intermittent, reduplicate, or imperceptible; (3.) Complete absence of cardiac impulse, and ^{at} an audible systolic sound; (4.) An excited or thumping action of the heart, associated with a very feeble radial pulse; (5.) Hurried respiration, whether “cerebral” or due to pulmonary lesion; (6.) Sleeplessness, associated with delirium, both of which are persistent; (7.) Severity of cerebral symptoms, and these symptoms coming on early; (8.) The occurrence of complete “coma-vigil;” (9.) Extreme contraction of the pupil; (10.) Extreme prostration occurring early; (11.) Muscular tremor, picking and catching at the bed-clothes, subsultus, and spasmodic twitchings of the muscles of the face; (12.) Urgent and protracted

hiccup; (13.) Rigidity of the muscles of the limbs, and squinting; (14.) Relaxation of the sphincters before the tenth day; (15.) Retention of urine; (16.) Tympanitis, with extreme nervous prostration; (17.) A dry, brown, hard, retracted, tremulous tongue; (18.) The more abundant and darker the eruption the greater the danger and severity of the case; (19.) Great lividity of the face and extremities, and a dusky erythematous condition of the skin on the dependent parts of the body; (20.) Continuous profuse sweating, coldness of the surface, cold breath, and a rapid weak pulse; (21.) A sudden diminution in the amount of the excretion of urea; (22.) The occurrence of blood or albumen in the urine before the tenth day, especially when associated with casts of the uriniferous tubes; (23.) Pulmonary hypostasis and bronchitis, pneumonia, gangrene of the lungs, convulsions, pyæmia, erysipelas, parotid swellings, inflammatory swellings, bed-sores, gangrene, renal disease, scurvy, the gouty diathesis.

2. Combinations of Symptoms or Phenomena which may be regarded as of favourable import.—(1.) A sudden fall in the frequency of the pulse; (2.) When a patient, after lying for days on his back, helpless and motionless, manages to turn himself round and sleep on his side, or if he is able to draw up his leg and rest it on the foot in the flexed position in the bed; (3.) Cases without rash, or in which the rash is scanty; (4.) When the excretion and elimination of urea and uric acid continue free and copious; (5.) Sudden cessation at the end of the second week of several of the unfavourable symptoms and phenomena; (6.) Diminution of the rapidity and increase in the strength of the pulse; (7.) A slight return of appetite, while the tongue becomes clean and moist at the edges; (8.) A diminution of the dusky tinge of the face, a less stupid appearance of the countenance, and a less injected state of the conjunctivæ, with signs of returning intelligence.

3. Modes of Fatal Termination.—(1.) Death during the primary fever may occur from syncope or from coma. In the former case the heart's action is enfeebled from paralysis or disease of its muscular tissue. In the mode of death by coma the blood has undergone such modifications as render it incapable of supporting the changes essential to existence. Its contamination seems mainly due to the admixture of urea and other products of the retrograde metamorphoses of tissue, and from the diminution and destruction or solution of its red corpuscles. (2.) Death is for the most part due to a combination of syncope and coma;

and, as a rule, the patient is quite unconscious for a considerable time prior to death. (3.) Death may occur from one of the many complications which happen before or after the cessation of the primary fever.

Morbid Anatomy.—The morbid anatomy of cases of typhus fever has been carefully investigated by Gerhard and Pennock, A. P. Steward, John Reid, Thomas Peacock, William Jenner, Felix Jacquot, Barrallier, and Murchison. All are agreed that there is no constant nor characteristic lesion; and they may be summed up generally as follows:—“A fluid condition of the blood; hyperæmia of the cerebral membranes and increase of intra-cranial fluid; bronchial catarrh and pulmonary hypostasis; softening of the heart, liver, spleen, and pancreas; hyperæmia and hypertrophy of the kidneys” (MURCHISON, p. 245).

Treatment of Typhus Fever.—Before considering the treatment of typhus fever, it is of the greatest importance to be aware of the changes which go on in the system during its progress. Dr. Parkes has observed the nature of these changes in a most conclusive manner. His observations are of great scientific interest, and of important practical bearing (“Gulstonian Lectures,” in *Medical Times and Gazette* for February 28, 1857). In an uncomplicated case of typhus fever the body loses flesh rapidly, owing not only to diminished ingress of food, but also to increased egress of bodily structures in the form of excretory products. The metamorphosis of tissue, as judged by the urine, is augmented. The only complete analysis of the urine in an uncomplicated and undoubted case of *typhus fever*, when no medicine whatever was given, is an analysis made and recorded by Dr. Parkes (*Urine in Disease*, p. 258). “The condition of the urine,” he writes, “was that of ordinary pyrexia. The water was lessened; the urea was increased one-fifth; the uric acid was in large amount, and spontaneously, or on the addition of an acid, deposited. The chlorides were entirely absent; there was no diarrhoea or sweating; the sulphuric acid was rather high; the phosphoric acid was not determined. The free acidity was very slight; and (differing from many pyrexiaë) the pigment and extractive matters were throughout in small amount. The urea continued large, and the chloride of sodium small in amount, for some days after the temperature had fallen to below the normal limit. The excretion of urea was remarkably regular in amount from day to day; for during ten febrile days its range was only

15 grains (1 gramme) below the mean of the ten days, and 20 grains ($1\frac{1}{2}$ gramme) above it. And this took place with great alterations of temperature. It then, as usual, fell during convalescence, and rose again to the healthy standard in three or four days. The chloride of sodium was clearly retained in this case, for there was constipation, and the skin was dry, so that none could have passed off by the intestines or surface." "It would seem also," he further observes, "that the urine in typhus is much more frequently albuminous than in typhoid fever."

In three cases of typhus fever associated with jaundice (which is extremely rare in typhus cases), Dr. Murchison examined the urine, which was also jaundiced. There was no re-action on testing for the bile acids; but in two of the cases *tyrosin* and *leucin* were found. In one of these cases the urine was almost devoid of urea. At the autopsies of two of them there was no derangement of the biliary ducts (*Path. Society*, Feb. 3, 1863).

The following inferences are drawn from the table given by Dr. Parkes:—

1. In spite of the many pints of fluid drank, a small quantity of water left the system by the kidneys and skin, and none at all by the bowels. This retention of water is not peculiar to typhus, and its cause is quite unknown.

2. The amount of urea was greatly increased. The normal amount of urea excreted by active men on good diet, between twenty and forty years of age, weighing 145 lbs., is 491 grains in twenty-four hours. A boy ill of typhus, aged seventeen, weighing not more than 129 lbs., excreted not less than 532 grains daily, although he was on fever diet, and taking scarcely any nitrogenous food.

3. The chloride of sodium is excreted in health at the rate of 180 grains daily. In this case of typhus fever it was present only in traces, the amount being too small to be determined. Like the water, this retention is common to the pyrexia.

4. Metamorphosis of tissue was more active by one-fourth daily.

General Indications for Treatment.—From most careful observations such as these, Dr. Parkes thus gives an outline of the principles upon which fevers are to be treated. The treatment of fever (and *typhus* and *typhoid* fevers are not exceptions) may be summed up as being a combination of measures to *reduce excessive heat*, to insure *proper excretion*, and to *act on the semi-paralyzed nerves*; and, as Dr. Murchison justly observes, "every

remedial agent which shall be found to promote the elimination of urea, without increasing the destructive metamorphosis of tissue, will deserve a trial in typhus" (l. c., p. 268).

To reduce heat and to regulate elimination are but secondary indications in the treatment of *typhus* fever, compared with the influence which must be exercised over the nervous system; and one of the greatest objects of therapeutics at the present day is to find substances which will act on the nerves and the blood, and restore them in some way to their normal action. (See page 155, ante.)

Special Indications for Treatment.—Our objects in the treatment of typhus fever should be,—(1.) To neutralize the poison and to correct the morbid state of the blood; (2.) To eliminate the poison and the products of the destructive metamorphosis of tissue; (3.) To reduce the temperature; (4.) To sustain the vital powers, and to obviate the tendency to death; (5.) To relieve the distressing symptoms; and (6.) To avert and subdue local complications (MURCHISON, p. 265).

1. In the belief that the morbid condition of the blood in typhus fever may be due to the presence of ammonia in some as yet unknown combination, the use of mineral acids has been recommended by many physicians. Murchison considers their beneficial effects in typhus as undoubted, and in this opinion he is confirmed by the experience of Huss of Stockholm, Haller of Vienna, and of Mackenzie, Chambers, and Richardson, in this country. Huss recommended phosphoric acid in doses of ten to fifteen drops every second hour, believing that the phosphorus exerts a special influence on the brain; but in the advanced stage, and especially if sweating, numerous petechiæ, or ecchymoses be present, he has recourse to sulphuric acid in doses of fifteen to twenty drops every hour or every second hour. Hydrochloric acid is preferred by Drs. Murchison, Richardson, Mackenzie, and Chambers. It may be given to the extent of one fluid ounce of the dilute acid, mixed in a quart of barley-water, sweetened with syrup of ginger, and flavoured with lemon peel. Dr. A. P. Stewart has used with advantage the *tinctura perchloridi ferri*, in doses of thirty minims every three hours. Dr. Murchison recommends *nitro-muriatic acid*. He prescribes twenty minims of *hydrochloric acid* with ten minims of *nitric acid* every three hours, each dose being diluted with the patient's

drink. But if the "typhoid state" is developed in a marked manner, *dilute sulphuric acid*, in doses of fifteen to twenty minims every three hours, in combination with *ether*, and small doses of *quinine*, are to be had recourse to as in either of the following formulæ:—

R. Acid. Hydrochlor. dil., ℥xx.; Acid. Nit. dil., ℥x.; Spt. Æther. Nit., ℥lx.; Liquor. Cinchonæ, ℥xxx.; Decoc. Scopar. comp., ʒi.; *misce*. A draught so composed may be administered every third hour.

Or, R. Quiniæ Sulph., gr. $\frac{1}{2}$; Acid. Sulph. dil., ℥xx. ad ℥xxx.; Æther. Sulph., ℥xv. ad ℥xxx.; Syrup. Aurant., ℥lx.; Decoc. Scopar. comp., ʒi.; *misce*. A draught so composed may be administered every third or fourth hour.

When the acids are cautiously administered in smaller doses, in conjunction with a few minims of solution of muriate of morphia, if the bowels be irritable, sweetened with syrup of orange peel and diluted with water, the draught is generally relished, and the tongue, from being dry, and hard, and brown, becomes moist and clean (PERRY).

2. To insure proper excretion and elimination in fever is much more difficult than to reduce temperature, which, for obvious reasons, it is not always wise to attempt the reduction.

Give Salts Perhaps the best general method to insure proper excretion is to supply the system with abundance of *alkaline salts*, which are not now given in the food, and to maintain the action of the kidneys, the bowels, and the skin. *Chloride of sodium*, the *alkaline salts of potash*, and probably also those of *soda*, tend to aid the formation of urea and its elimination. In the use of *nitrate of potash* and of *iodide of potassium*, which are not natural constituents of the frame, Dr. Parkes has observed that, at the first employment of these, there is often a marked lessening of excretion, as if the chemical processes then going on in the body had been interfered with, for afterwards the elimination again increases, as if the system had accommodated itself to the remedy.

Purgatives tend to insure a proper excretion, probably by removing from the blood some of the abnormal products formed in fever. The great relief which sometimes follows their use, as well as the fall of temperature, seems to show this. Where there is retention of urea, they aid its elimination, because we know that urea passes off sometimes by the mucous membrane of the

stomach and bowels. The patient should be allowed to drink freely of water; and five grains of the *nitrate of potash* may be given with each dose of the nitro-muriatic acid already mentioned. Dr. Murchison recommends *nitre whey*, prepared by boiling ℥ii. of nitre in a pint of milk, and straining; or a drink prepared by dissolving ℥i. to ℥ii. of the *bitartrate of potash* in a pint of boiling water, and flavoured with lemon peel and sugar; but if the patient be very prostrate, or if the bowels be relaxed, *nitric ether* is to be substituted for the *nitrate of potash*.

Tea and coffee have been recommended in the stupor of typhus; and it is probable, as Dr. Parkes has shown, that their good effects are due to their power of eliminating the urea already formed in the blood. The coffee may be given as an extract, or as a strong infusion of the powdered berry made in the ordinary way. Tea has been recommended as an infusion of the green tea leaf. As beverages or common drinks in fever, both tea and coffee have been found to relieve the headache, the pulse becoming fuller and stronger under their use. Böcker, L. Lehmann, and Hammond, all agree in showing that in health they greatly lessen the urea (Parkes *On the Urine*, p. 76). With respect to *chloride of sodium*, Dr. Murchison recommends that large quantities of this salt should be given with the beef tea.

The action of the bowels is to be maintained by emetics and laxatives. In the first instance, if the patient is seen early—i. e., before the sixth day—an emetic of ipecacuanha (one scruple), and of antimony (one grain), or of carbonate of ammonia (two scruples), in place of the antimony, is to be administered. If the bowels remain confined after the emetic, a mild laxative of rhubarb and calomel, or of castor oil, is to be given; and failing these, or in place of them, a simple enema is to be administered (MURCHISON, p. 269). The advantages of emetics are, that they relieve the patient to some extent by mitigating or removing headache and general pains. They also reduce the temperature, abate thirst, and quiet gastric disturbance.

Emetics, however, are contra-indicated if the patients are unusually weak, or if the disease has advanced beyond the first week. Laxatives and enemata, however, ought to be repeated daily, if required, so as to secure a motion of the bowels once a day. In this respect the treatment is different from the treatment which ought to obtain in typhoid fever, as already mentioned.

Excrementitious matters in the intestines must be removed by gentle aperients. The dark offensive matters accumulated in the intestinal canal in typhus fever may have a secondary deleterious effect on the system if they are allowed to remain. Purging, however, is to be avoided, and *fresh-made* compound rhubarb pill mass, which *tends to stimulate the peristaltic action of the intestines*, is as good a medicine as can be given, followed, if necessary, or alternated, by a small dose of castor oil, or by a simple enema.

Diaphoresis is not to be encouraged beyond the insensible transpiration of the skin; and to remove which the wholesome detergent of tepid water sponging is most beneficial. It ought to be used twice or three times daily, and quantities of *Condy's fluid* or of *muratic acid* (ʒi. ad Oj.) may be mixed with the tepid water (MURCHISON). The measure is a good one in a hygienic point of view, and it contributes—

3. To reduce temperature, for which the external application of cold water was once practised to an extreme degree by Currie, as originally recommended by Dr. Robert Jackson. In health such an application as that of cold water has a great effect in reducing temperature, and tends to increase metamorphosis (LEHMANN, SANDERSON).

4. The vital powers are to be sustained by food in the first instance. For this purpose, *nourishment ought to be given often, and at stated intervals—at least once every three or four hours after the fourth day of the fever.* Even if the patient is asleep, or seems to be so, he must be roused at these stated intervals (not oftener) to take his food or his stimulants. But if, towards the period of the crisis, the patient appears to be in a sound sleep, he ought not to be disturbed. The indications for treatment just described apply to the earlier stages of the fever, up till about the fourteenth day.

Alcohol in small quantities, as well as tea, coffee, and other substances, have a directly stimulant action on the nervous system, and on the organs of circulation; at the same time, they diminish the metamorphosis of the tissue-elements. Few remedies, however, require more discrimination in their use; and the following guides for their administration are compiled from the careful observations of Dr. Murchison (l. c., p. 269):—

1. Wine is not usually required during the first five or six days of the illness, but most cases require some stimulants during the second week; and, as a rule, the physician may find it

necessary to begin to administer stimulants about the seventh or eighth day.

2. The indications for the administration of alcoholic stimulants are mainly derived from the state of the organs of circulation; and the profession is indebted to Dr. Stokes (1839) for pointing out the importance of cardiac and radial pulses as guides for the use of alcohol in fever.

These indications are,—

(a.) Extreme softness or extreme hardness and compressibility of the pulse. An irregular, intermitting, or imperceptible pulse more imperatively demands stimulants than a merely rapid pulse. So also an abnormally slow pulse—*e. g.*, 40 to 60—is a stronger indication for stimulation than a quick pulse.

(b.) When the cardiac impulse becomes weak, and when the first sound is impaired or absent, a liberal allowance of stimulants is demanded: and in every case where there are doubts as to the propriety of giving stimulants, the heart must be examined with the hand and with the stethoscope, because the state of the pulse alone is not sufficient to judge from. The impulse may be found to diminish progressively from the fifth or sixth day to the termination of the disease; and for several days prior to death or recovery it may be entirely absent. The systolic sound of the heart becomes daily more feeble, and ultimately may be quite inaudible, leaving the second sound clear and distinct; and before the first sound is altogether lost, it may become so short that it is difficult to distinguish it from the second sound. If the action of the heart be rapid, its sounds may thus come to resemble closely those of the *foetus in utero*. A violently excited heart all through the disease, with cold surface, cold breath, and feeble pulse, demands wine from the first; but even with its judicious use the prognosis in such cases is extremely doubtful (STOKES, GRAVES, MURCHISON).

Other indications for stimulants may be stated as follow:—

(c.) If by raising the patient to his semi-erect position a tendency to syncope is induced, or great prostration is manifest, with diminished strength and volume of the pulse, then stimulation must be commenced.

(d.) The darker and more copious the eruption, the more is the necessity for stimulants, especially if petechiæ are numerous.

(e.) Profuse perspiration, with no improvement in the general symptoms, requires an increased supply of stimulants.

(f.) Coldness of the extremities, stupor, low delirium, tremor, subsultus, involuntary evacuations—symptoms generally of the “typhoid state”—are indications for the liberal administration of alcohol; but the propriety of giving stimulants in delirium depends on the state of the pulse. If, on the trial of stimulants, the patient becomes tranquil, they do good, and may be continued; if the reverse, their use must be suspended.

(g.) A dry brown tongue is an indication for wine or brandy, and if it becomes clean and moist at the edges under the use of either, such stimulation is beneficial.

(h.) Complications, as a rule, increase the necessity for stimulation; and large quantities of stimulants are called for if *pyæmia*, *erysipelas*, *bronchitis*, *pulmonary hypostasis*, *pneumonia*, *inflammatory swellings*, *bed-sores*, or *local gangrene* should supervene.

(i.) Persons who have led intemperate lives, and old persons, require stimulants early in the fever, and in large quantities.

The effects of alcoholic stimulation require to be most carefully watched throughout the whole period of their administration. Four ounces of wine in the twenty-four hours is enough to begin with; for if the blood be overloaded with the products of alcoholic ingestion, further alcoholic stimulation will lead to increased contamination, and it is rare that more than eight ounces of brandy in twenty-four hours are necessary.

There are differences in the demand for stimuli in the typhus of different countries, and in the fever of different epidemics. Dr. Wood tells us that in America cases requiring wine or brandy are extremely rare. Dr. Stokes says that the *typhus* in Ireland demands large quantities of wine. In Scotland, also, wine is the great mainstay in the treatment of *typhus fever*, requiring often to be administered largely.

Port, Sherry, Marsala, Madeira, brandy, gin, or whisky, possess no peculiar advantages apart from the alcohol contained in each. Spirits contain from fifty to sixty per cent. of alcohol, Sherry and Port from seventeen to twenty-four per cent., and malt liquors from six to eight per cent. Two fluid ounces of spirit will thus be equal to five or six of wine, and spirits ought to be given diluted; and if the prostration is great, and when the skin is cold, and covered with perspiration, the best stimulant is brandy or whisky punch, given as hot as it can be taken, in small quantities at a time, frequently repeated. In urgent cases stimulants ought to be given every hour; and, as a rule, a larger

quantity will be required during the night and early morning than in the daytime, for it is usually towards morning that temperature tends to get low, and the vital powers are at their lowest ebb (MURCHISON). At the same time it must ever be remembered, as Dr. Jenner justly observes, that "in no disease is the advantage of refraining from meddling more clearly displayed than in typhus fever; and in no disease is the prompt use of powerful remedies more clearly indicated. It is in determining when to act, and when to do nothing, that the skill of the physician as a curer of disease, in the case of fever, is shown. Interfere by depletion or by stimulation when nothing should be done, and the patient is lost, who, if it had not been for you, would have been safe. Refrain from depletion or withhold stimulants when the one or the other is required, and the patient sinks into that grave from which judicious treatment might have saved him."

A large well-ventilated apartment, fresh air, a cool, but not a cold atmosphere, quiet, abstinence from solids, and a free supply of water, milk and water, coffee, weak broth, beef tea, according to the discretion of the physician, are the conditions and remedies on which a large majority of cases will recover. But the patient must be constantly and carefully watched, and there is no disease where the attentions of a well-instructed nurse are more demanded; and there is no class of patients in hospital so apt to be neglected by the attendants, especially as to the regular administration of the remedies prescribed. It is not uncommon to find that the wine allotted for the day has been administered at a draught, when it ought to have been given in small quantities at regular intervals, with care and watchfulness. How often do we see almost hopeless cases recover under the careful nursing of an intelligent person, regulated by the dictates of common sense and conscientious solicitude, guided by the judicious directions of a physician who knows well the nature of the disease with which he has to deal! The nurse ought to note down the hours at which food or medicine has been given, or any remarkable change in the symptoms. She might also, if she were instructed, take observations with the thermometer, for the information of the physician at each visit.

Dr. Murchison recommends that, in urgent cases, food and alcoholic stimulants must be persisted with as long as the patient

is able to swallow; and even when he can no longer swallow, the case is not to be given up; for he has seen cases where life appeared to be saved by frequent enemata of beef tea and brandy after the patient had ceased to take anything by the mouth.

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Of special symptoms which call for relief, the most urgent is generally headache. If headache should persist after delirium sets in, with a rapid pulse (*e. g.*, 120), attended with nausea, some saline effervescing mixture, with four drops of hydrocyanic acid, may be given every six hours. In the persistence of headache, dry cupping, such as has been recommended by Dr. Sieveking, might furnish an aid to guide the treatment, by determining whether it may not depend upon repletion or upon emptiness of the cranial vessels. When applied to the nape of the neck, dry cupping may afford relief, if repletion has to do with the continuance of headache. Under such circumstances the face is generally flushed, the conjunctivæ red, and the skin dry and hot. If the dry cupping does not relieve such symptoms, the hair must be shaved off the head, and the scalp covered with crushed ice enclosed in a bullock's bladder, or recourse may be had to the cold affusion. The application of cold water is best effected by bringing the patient's head over a basin at the edge of the bed, and having a vessel arranged so that the cold water (at 40° or 50° Fahr.) may drip continuously from a height of two or three feet upon the head (MURCHISON). A skein of worsted arranged in the water, with the ends overhanging the basin, will maintain a constant flow of water from the basin, and which may be directed to fall upon the scalp. Dr. Murchison recommends that in young subjects two or four leeches may be applied to the temples; and in aged or infirm persons warm fomentations to the head are advisable (GRAVES and MURCHISON). But if anæmia is the cause of the headache, as may be suspected from the state of the vascular system, then stimuli are called for. Four to six ounces of wine may be given in divided doses during the day and night of twenty-four hours. If the pulse continues to get weaker, the wine must be increased.

The headache of typhus naturally abates about the eighth day; but it is sometimes rendered worse by sleeplessness; and if the remedies for the headache do not relieve it, nor tend to induce sleep, then opiates may be given, combined with antimony, if

the skin be dry and hot and the pulse of good strength. Dr. Murchison thinks that the employment of opium in typhus is more dreaded than it ought to be. The dose of opium should be given about 9 P.M., followed in two hours by half the dose if the patient does not sleep. The form of the opiate and dose may be ten to twenty minims of Battley's solution of opium, or fifteen minims of the solution of the *bimeconate of morphia*, or five grains of the *opium pill* of the *British Pharmacopœia*. Dr. Murchison teaches us to distinguish two forms of delirium as a guide to the administration of opium, combined with antimony in the one form, and with ethereal stimulants in the other. When the condition of the patient approaches more to that of *delirium ferox*, the cardiac and radial pulses being of good strength, after trying the cold affusion, and remedies already mentioned, then opium combined with antimony ought to be given without delay, as in the following prescription:—

R. Liq. Opii. Sedat. (Battley's), ℥lx. ; Antim. Tart., gr. i. ad gr. ii. ; Aquæ Camph., ℥vi. ; *misce*. A large spoonful of this mixture is to be given every hour until sleep is induced.

On the other hand, if the delirium approaches in its character that of *delirium tremens*, the radial pulse is usually quick and feeble, the cardiac impulse diminished, and the first sound of the heart more or less inaudible, then the opium must be combined with alcoholic or other stimulants, the amount being regulated by the state of the pulse and heart. Dr. Murchison suggests the following prescription:—

R. Liq. Op. Sed. (Battley's), ℥ss. ; Spt. Ætheris, ℥lx. ; Aquæ Camph., ad ℥iii. ; *misce*. Commence by giving two table-spoonfuls of this mixture, and repeat it every hour till sleep is obtained.

Or opium to the amount of half a grain may be combined with three grains of camphor in a pill, and such a pill may be repeated, if necessary, every two hours.

Cases requiring such treatment ought to be seen at least three or four times daily. If dyspnoea is urgent, and lividity of the face betoken pulmonary lesion, defective arterialization of the blood, and venous congestion of the brain, opium in any form must be withheld; and it must likewise be discontinued if any tendency to stupor supervene, or if there be any marked contraction of the pupil—*e. g.*, “the pin-hole pupil” of Dr. Graves. This

physician proposed the use of *belladonna* in such cases, and he, as well as Dr. Benjamin Bell and Dr. Murchison, bear their united testimony to its usefulness. Dr. Graves prescribed it as follows:—

R. Ext. Belladonnæ, gr. ¹/₄; Ext. Hyoscyami, gr. vi.; Pil. Hydrar., gr. xxx.; *misce*. This mass being divided into six pills, one may be given every three hours; or it may be given in the form of a draught, in the following prescription:—R. Ext. Belladonnæ, gr. i.; Pulv. Moschi, gr. x.; Mucilaginis Acaciæ et Syrup. Aurant., ā ā ℥ cxx.; Aquæ Camph., ℥ ss.; *misce*. A draught of this composition may be given every six hours.

Dr. Murchison considers that musk and camphor are stimulants of very great value, which have fallen into unmerited neglect. Camphor may be given in emulsion in doses of *five* grains every two hours; or in the form of an enema in doses of a scruple. Huss and Graves also bear testimony to the good effects of these remedies. In a case of complete sleeplessness Dr. Graves gave the following combination of these medicines with the best results:—

R. Antim. Tart., gr. ss.; Pulv. Moschi, gr. x.; Camphor, gr. v.; Tinct. Opii., ℥ x.; Aquæ dil., ℥ i.; *misce*. A similar draught may be given every two hours; and after the third dose the patient will generally fall into a quiet sleep.

When there is danger of stupor passing into profound coma, Dr. Murchison has seen the best effects result from a small cupful of a strong infusion of coffee given every three or four hours, employing at the same time such measures as have a derivative action on the kidneys, *e.g.*, dry cupping; mustard poultices to the loins, wet compresses of thickly folded flannel, wrung out of hot water, passed round the loins, and covered with a piece of waterproof cloth, retained in its place by a bandage or a towel. These remedial agents are all the more necessary if the urine contain either blood or albumen. At the same time free evacuation from the bowels should be secured by a purgative, or by a turpentine enema. If the lethargic state supervenes early, and before there is great exhaustion, the douche has been found to be of great service as a stimulant, provided there be considerable elevation of temperature, and little irritability of the nervous system (TODD, ARMITAGE, MURCHISON).

The region of the bladder should be examined by the physician at least two or three times daily, by manipulation and percussion; and if there be the slightest doubt as to its containing urine, the catheter must be introduced. MB

Origin and Propagation of Typhus Fever.—It is yet uncertain whether great overcrowding and vitiation of air by the organic impurities emanating from the respiratory and other functions will absolutely generate typhus fever *de novo*. In all the English wars (for “typhus fever is a disease as old as the disputes of nations”) there has always been plenty of typhus poison waiting for favourable conditions to assume activity. This arose from the peculiar system of recruiting. Commissions or commands of regiments were wont to be given to those who collected a certain number of men. Every low purlieu, every infamous haunt, every jail even, used to be ransacked for recruits. Wherever these men went they carried typhus, at that time the constant scourge of our towns and our jails; and complaints of the introduction of typhus fever from this source are frequently found in the writings of army surgeons of the last century. In connection with this point, Dr. Donald Munro, in 1764, gives the following caution:—“That particular regard be paid to those soldiers picked up in the streets, or who have been taken out of the Savoy or other jails. All dirty rags from such people should be thrown away or burnt,” (DR. PARKES, l. c.) There is now ample proof that typhus fever may be communicated *by fomites* adhering to apartments, articles of clothing, and the like; and, provided fresh air be excluded, it is known that such articles will retain the poison for a very long time. Herein lies a fallacy which pervades the argument from cases to prove the generation of the disease *de novo*. The poison may be said (like that of small-pox) to be constantly in existence. Dr. Murchison quotes some striking instances of the propagation of typhus fever by *fomites*. For example, he refers to the instance related by Foderé, in which the soldiers of the French army, during their retreat from Italy in 1799, communicated fever to the inhabitants of towns and villages where they halted on their route, although the army was not attacked by fever, and soldiers travelling *singly* did not communicate the disease. But as he omits to connect this with the fact that typhus prevailed to a great extent in the towns they besieged, and in some instances obtained possession of, the

source of the fomites is not made apparent, and therefore in my last edition I was led to misrepresent this instance given by Dr. Murchison, and to put it forth as an example of generation *de novo* (See p. 87 of his work *On Continued Fevers*). He quotes, however, the recent instance of the Egyptian vessel, the "Scheah Gehald," at Liverpool, the crew of which disseminated the poison of typhus by their clothes and persons, although, as he says, they had not the disease themselves. But this is an error. The careful investigation made by Dr. Parkes into the history of this epidemic on board the Egyptian ship clearly shows that the crew suffered from typhus fever (*Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1860, p. 359). The facts of the case have been curiously confused; but the following statement, from the above and other sources, may be relied on:—A number of men (476, chiefly Arabs) were shipped on board the "Scheah Gehald" at Alexandria, to proceed to Liverpool to navigate back a man-of-war then in that port. The weather was cold and stormy, the hatches were battened down during a lengthened voyage of thirty-two days from Malta; and the men, unaccustomed to the rigor of a Northern winter, and not provided with suitable clothing, crowded below for warmth and shelter. Even they whose turn it was for duty had to be driven up on deck. They were extremely crowded on board, and the space below deck was quite insufficient for so large a number (for the crews of two vessels were on board); and there was no attempt to promote ventilation. The persons and clothing of the men were filthy in the extreme. The space between the decks soon became intolerable from filth; for many of the men, being landsmen, were sea-sick on the voyage, and they discharged the contents of their stomachs and bowels in all parts of the ship, which, on arriving at Liverpool, was so offensive that it had to be sunk in the graving dock. Moreover, the rations served to the men were much below the proper standard as regards quantity. Several deaths occurred on the voyage; and although the captain denied the existence of fever and the occurrence of deaths, his statements are quite untrustworthy, for on arrival in Liverpool thirty-two men had to be sent to the Southern Hospital. Two died soon after admission, and their disease was returned as dysentery; but Mr. Pemberton, on whom the duty of receiving and treating the patients at first fell, was convinced that he had

some kind of fever before him in the persons of these sick Arabs. He called the disease "febris;" and in writing to a friend expressed his belief that it was a "jail fever." The heat of skin, the sordes on the teeth, and the marked symptoms of stupor in some cases and furious delirium in others, led him to this conclusion, although he could not see the eruption on the dusky skins of the Egyptians; and perhaps, as he had never seen a case of typhus fever before, he might not have recognized the eruption, as Dr. Parkes suggests, even if present. This diagnosis, however, was made at once by Mr. Pemberton, and before fever had been communicated to any residents in the hospital. It is impossible now to ascertain how many of the thirty-two Egyptians had this fever; but five had marked, and several others had slighter symptoms. Many of the patients (typhus and others) had dysentery, and several were frost-bitten.

Indubitable typhus fever, with a well-marked rash, was communicated by these men, and by Mr. Pemberton himself (who had a well-marked rash), to another medical officer, and to two nurses, a porter, and some patients. The chaplain, also, who slept out of the hospital, but visited the sick men, was attacked, and died in twelve days. In all, nineteen persons contracted typhus in the Southern Hospital, three on board the ship after she came to Liverpool, and three at the Liverpool baths. Six died of these twenty-five persons. "No single link of evidence," says Dr. Parkes, "is wanting here to show that typhus prevailed on board the ship, and that typhus patients admitted from the ship into hospital communicated the disease to a number of other persons. The idea that the Egyptians suffered only from dysentery, and that in some remarkable way a specific disease like typhus arose out of this dysentery, does not appear to have the slightest foundation. To urge such an hypothesis, in the face of the simple facts above noted, is to ignore all evidence, and to render the progress of medical science impossible. Cases of typhus were not only communicated to residents in the hospital, but to persons who boarded the ship, and to three attendants at the public baths, to which more than 200 of the crew were sent. Some of these men were sick, though they were not known to have typhus. They carried typhus, however, in some way—perhaps in their clothes—and communicated it to the attendants. The remaining crew (350) of the 'Scheah Gehald' were sent to

Alexandria on board the 'Voyageur de la Mer.' The people of Liverpool were probably so glad to get rid of them, that they did not take the trouble to see that the typhus fever had been eradicated, and several of the men were sent at once from the Southern Hospital. The 'Voyageur de la Mer' lost some men on the passage, and landed several at Falmouth, and some, with unequivocal typhus, at Malta; and of thirteen Englishmen who were in her, six took the disease."

"The case of this Egyptian vessel," continues Dr. Parkes, "afforded almost the best opportunity seen in this generation for the investigation of the important question of the spontaneous generation of typhus. The opportunity was, however, lost. That all the circumstances which have been supposed to be capable of calling into existence the specific poison of typhus were present in this foul and filthy ship is clear; but every one who reads all the published statements will at once perceive that one link of the chain of evidence is wanting, and that it has *not* been proved that some of the crew were not ill with typhus when they embarked at Alexandria, or became ill within the incubative period. On the contrary, the interpreter informed Mr. Pemberton that some of the men were sick when they came on board. It can never now be ascertained whether there were such cases or not, and the history of the outbreak at Liverpool affords another instance of the loss of a great opportunity for definitely setting at rest a most important question." The case of the "Scheah Gehald" now assumes exactly the same aspect as many instances historically quoted as examples of generation *de novo*—namely, that however plausible may seem the probability, there is *no proof* that typhus fever arose *de novo*. Seeing that such is the state of the question as to the origin of typhus—that it is exactly in the same state as our knowledge regarding the origin of small-pox or of typhoid fever—that it has been in existence from the earliest periods of the world's history—that it is undoubtedly propagated from pre-existing *foci*, and by continuous succession, the immediate direction of investigation ought to bear especially on the following points, namely:—How long can the typhus poison exist or be maintained in a condition fit to assume activity under favourable circumstances? What is the distance at which it is potent? Has temperature any influence upon it? What are the conditions or combination of circumstances more or

less essential to the development and propagation of the typhus poison?

The fact that typhus fever is contagious is based on evidence which shows,—(1.) That, when typhus commences in a house or district, it often spreads with great rapidity; (2.) That the prevalence of typhus in single houses, or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick; (3.) That persons in comfortable circumstances, and living in localities where the disease is unknown, are attacked on visiting infected persons at a distance; (4.) That typhus is often imported by infected persons into localities previously free from it; (lastly), That its contagious nature is indicated from the success attending the measures taken to prevent its propagation, more especially the early removal of the sick. Dr. Murchison fully illustrates by examples all of these statements.

The specific poison seems capable of transmission in various ways; but many circumstances seem to point to the cutaneous and pulmonary exhalations of the sick as the media which convey the specific poison from the diseased to the healthy. It is thus conveyed through the air, or by fomites. That particles of organic matter are constantly floating in the air no one can doubt who has seen the ingenious contrivance of M. Pouchet put in practice, and the substances so suspended in the atmosphere collected by drawing a current of air through a funnel with a very small opening. Immediately below the opening the covering glass of a microscope slide is placed, with a drop of glycerine spread over it. Upon this the current of air impinges, and any solid or corpuscular bodies floating in the air may be in this way arrested and examined with the microscope. Dr. Parkes has detected by this method unequivocal epithelium-cells in several instances; and Eiselt, in a ward containing thirty-three children with acute purulent ophthalmia, was able to detect pus-cells floating in the air (*Army Med. Dep. Sanit. Report* for 1860, p. 346; also *Comptes Rend.*, 1861; and *Med. Times and Gazette*, 1861).

Such material particles, capable of conveying the specific poison of a disease such as typhus, are thus inhaled or swallowed, and so they find admission into the bodies of the healthy, to exercise their morbid influence on the blood. A peculiar pungent odour

emanates from the typhus fever patient. It is especially obvious from the breath, and from the skin on turning down the bed-clothes.

There is no evidence to show the extent of space through which the typhus poison can be transmitted through the air. From some observations it would seem that the contagious influence of typhus is confined to a narrower sphere than that of small-pox. Dr. Murchison concludes that, "if a patient be placed in a large well-ventilated apartment, the attendants incur little risk, and the other residents in the same house none whatever."

"There are likewise no grounds for the popular belief that typhus may be propagated through the atmosphere from a fever hospital to the houses in its neighbourhood. On the other hand, medical attendants who auscultate typhus patients, or who inhale their concentrated exhalations from under the bed-clothes, run no small danger, and the danger is always increased or diminished in proportion to the supply of fresh air" (MURCHISON, l. c., p. 80). There are also good grounds for believing that typhus fever may be communicated, and even carried a great way, by fomites, or by articles of clothing strongly impregnated with the specific poison; and, provided fresh air be excluded, the efficiency of the poison may be maintained for a long time. "Complaints of the introduction of typhus from this source are frequently found in the writings of army surgeons of the last century. Typhus was several times carried to the West Indies, and even there prevailed apparently to some extent" (Parkes *On the Causes of Sickness in English Wars*). The poison may also adhere to the walls of dwellings, to beams of wood, and to articles of furniture. Dr. Murchison quotes an account by Pringle of twenty-three persons being employed in refitting old tents in which typhus patients had lain; and seventeen of these persons died of the infection. He also refers to an observation of Lind, who mentions several instances in which infected ships continued to impart the disease long after the sick had been removed. Similar cases are recorded by Jacquot respecting the Crimean typhus.

Nurses and other attendants in fever hospitals are well aware of the danger of contracting typhus from infected clothes, and from cleaning the bedding of the sick; and in some instances they are in the habit of "measuring the amount of danger by the

badness of the smell." Thus they are liable to contract typhus fever without having had any direct communication with the sick. With regard to the kind of clothing most apt to retain and convey the specific poison, woollen textures are found to be the most dangerous. Haller of Vienna has made experiments on this point. He observes that *dark-coloured materials of clothing* are more apt to absorb the contagion of typhus, and to convey it to other individuals, than those which are light-coloured. He found that, among troops wearing dark-coloured uniforms, it more frequently happened that new cases of typhus entered the hospital after a convalescent patient joined his corps than those wearing light or white uniforms. The fact has been often observed, that in dissecting-rooms dark clothes acquired the cadaveric odour sooner, and were deprived of it less readily than light ones; and he ascertained by experiments that the absorption of odours is regulated by the laws which govern the absorption of light. Haller also found that the specific poison of typhus fever is *lighter than atmospheric air*. When the under storeys of an hospital were filled with typhus patients, those in the upper storeys were always observed to become infected when there was a communication between the air of the two storeys. On the other hand, when only the upper storeys contained cases of typhus, the patients in the under part of the house enjoyed perfect immunity (*Edin. Med. and Surg. Journal*, 1853). Dr. Murchison has observed that, if the poison be very concentrated, the length of the period of exposure sufficient to contract the disease is very brief—not more than a few minutes; and the latent period during which it remains in the body, without betraying its presence in any way, has been very variously estimated. Nine days is the result of Dr. Murchison's observations. Instances, however, are not uncommon in which the disease manifests itself almost instantaneously after exposure to the poison. In such cases these extremely susceptible persons are generally conscious of the peculiar and offensive pungent odour emanating from the beds or bodies of the sick. They are generally then immediately seized with prostration, nausea, rigors, and headache, followed by the regular development of the disease. Such persons are thus almost conscious of the moment at which the poison entered their system. On the other hand, the length of time between exposure and attack may be greatly prolonged. In my own case, I was three months in daily and close

attendance in the fever wards of the Dundee Infirmary, for many hours, on cases of typhus fever, before taking the disease. In such cases, however, of prolonged exposure, it is probable that the constitution may be susceptible at some periods rather than at others; but of the conditions of such susceptibility nothing is known.

Opinions vary as to the stage of the disease at which the typhus poison is most powerful. Some consider that it is so during the period of the eruption—others that it is so during the period of convalescence. Dr. Perry was of this latter opinion, and Dr. Murchison's observations lead him to confirm the opinion of Perry; but he is inclined to think that the disease is really most apt to propagate itself from the end of the first week up to convalescence, when the peculiar odour from the skin and lungs is the strongest.

The conditions essential to the propagation of the specific poison of typhus fever are mainly as follows:—(1.) Overcrowding, co-existing with deficient ventilation; (2.) Personal squalor, and filthy apparel saturated with cutaneous exhalations; (3.) A deteriorated state of the constitution, such as may result from protracted starvation, scurvy, and other debilitating causes; (4.) A moderate temperature. Dry heat is a powerful disinfectant.

RELAPSING FEVER.

LATIN, *Febris recidiva*; FRENCH, *Typhoïde à rechutes*; GERMAN, *Typhus recurrens*; ITALIAN, *Tifo ricadente*.

Definition.—A continued fever, having a very abrupt invasion and short duration, marked by rigors, chilliness, and severe headache, vomiting, and often jaundice; a white moist tongue, epigastric tenderness, confined bowels, enlarged liver and spleen, high-coloured urine, a frequent, full, and often bounding pulse, pains in the back and limbs, restlessness, and occasionally delirium. These symptoms abruptly terminate by an exceedingly copious perspiration between the fifth and the eighth day; and after a complete apyretic interval (during which the patient may be so well as to get up and walk about), an abrupt relapse supervenes on the FOURTEENTH day from the first commencement. The relapse runs a similar course to that of the primary paroxysm, and terminates between the third and the eighth day. In some cases a second, third, fourth, and even fifth relapse may

occur. *Death is apt to happen from sudden syncope, especially after the excessive perspiration; or from suppression of urine and coma. No constant eruption and no specific lesion are associated with this fever.*

Pathology.—The name by which this disease is known is derived from one of the most constant and striking peculiarities of the fever. It has been also described under the various names of “five” or “seven-day fever,” “seventeen-day fever,” “bilious remittent fever,” and “bilious relapsing fever,” “mild yellow fever,” “synocha,” “short fever,” and “short relapsing fever.” Epidemics of this form of fever have been recognized to prevail on different occasions since 1739. In Dublin it prevailed at that time and in several subsequent years. Sometimes it has been described as a variety of a well-known form of fever, and at other times as a new disease.

In Scotland in 1817-18 this fever was clinically recognized and described by Drs. Christison and Welsh; and when it re-appeared as an epidemic in Edinburgh and Leith in 1843, Dr. Christison had no difficulty in again recognizing it. About this time it also appeared in Glasgow as an epidemic about a month before its outbreak in Edinburgh; and subsequently it became prevalent in Dundee and other large towns in Scotland. It was observed with great accuracy, and its phenomena were recorded in the medical journals of the period, by Drs. Craigie, Alison, Arrott, Henderson, Douglas, Jackson, Mackenzie, Cormack, and Wardell. It formed a part of the fever epidemic of Ireland in 1817-18-19, described by Barker and Cheyne; and it had been prevalent in Ireland for many years. Epidemics of it were described by Rutty, in his *Chronological History of the Diseases of Dublin*, as early as 1739 and 1741. In most of the periods of epidemic fever referred to, the commencement of the epidemic was characterized by the greater preponderance of cases of relapsing fever; and as the epidemic advanced, the number of cases of relapsing fever gave place to a preponderance of typhus cases (STEELE, R. PATERSON, ORMEROD, MURCHISON). In 1847 it became again epidemic in Glasgow, Edinburgh, and the large manufacturing towns of Scotland, as well as in London, when it was carefully described by Dr. Jenner, who, moreover, shows that its characters have remained constant since they were first described by British physicians. During the same year it prevailed in some parts of

the Continent, and more especially in the Prussian province of Upper Silesia, and in some other parts of Germany. There it has been described by Virchow, Bärensprung, Dümmler, and Suchanek. These observers, however, did not know or recognize the fever so well and precisely described by the Scotch physicians; and, indeed, Dr. Parkes was the first to indicate, in his admirable paper on "The Diagnosis of Fevers," already noticed, that the epidemics these German physicians described were mainly made up of the relapsing fever. This fever evidently formed the great bulk of the cases. Yet, although its characters are thus so striking that the most superficial observer could not fail to recognize them, the German systematic writers (except Virchow) make no allusion to relapsing fever as a separate and distinct disease; and even those who observed the fever in Germany failed to draw that obvious inference to which the Scotch physicians unanimously came—namely, that *relapsing fever* is a disease altogether distinct from *typhus* and from *typhoid fever*. If it is not so, "we know not," as Dr. Parkes observes, "that any medical evidence whatever can be relied upon."

In the summer of 1855 it prevailed, after the hardships and privations of the preceding winter, among the British troops in the Crimea, where it was recognized and described by Dr. Lyons. It has not been observed in France, nor in any other part of the continent of Europe.

The observations of Dubois, Austin Flint, and others, leave no doubt that relapsing fever was seen in New York, Buffalo, and other parts of North America, in 1847 and 1848; but Dr. Murchison is of opinion that all the cases are traceable to Irish immigrants, and that there is no good ground for believing that the disease is indigenous in America. It has been well described by Dr. Wood, of Pennsylvania, from the writings of the physicians already mentioned. In India and in all tropical countries it is as yet unknown.

Since the epidemic of 1847 and 1848, Dr. Murchison writes that relapsing fever has been gradually disappearing; and for the seven or eight years previous to 1863 not one case has been observed in the hospitals of Edinburgh, Glasgow, or London. Professor W. T. Gairdner has not seen or heard of a single case at Edinburgh since 1855; and, according to Drs. Lyons and M'Ewen, true relapsing fever has of late years been a rare disease in Ireland.

Printed spec. in London Thralter 69-70
As Fever gives no immunity from typhus or typhoid

Like other continued fevers, its specific cause is unknown; but it selects its victims from the poor and ill-fed, who live miserably, in crowded, filthy, ill-ventilated apartments, rather than from the wealthy and well-fed, who live in comfort and in well-aired abodes. Its poison appears to be of a specific kind, and the phenomena of the fever are very different from those of *typhus* and *typhoid fever*. Patients recovering from either *typhus fever* or *typhoid fever* may catch, by contagion, the *relapsing fever*, while patients convalescent from *relapsing fever* may also take either of the forms of *continued fever* already described. It has been supposed by some (DR. CORMACK) to be identical with the malarious form of *yellow fever*; but there is not sufficient evidence to establish the point. It seems more nearly to approach in its nature some forms of remittent fever, on account of the repetition of the rigors, often at regular daily periods, for two or three days. The marked periodicity of its relapses, which "come on like a fit of ague almost to an hour" (DR. R. PATERSON), and the enlargement of the spleen to a greater extent than in any other form of fever (JENNER), point also to a malarious origin. On the other hand, epidemics of relapsing fever, as Murchison shows, appear to commence, progress, and decline quite irrespectively of the season of the year.

The evidence that a specific poison exists and is formed in cases of relapsing fever, and when so formed is communicable from the sick to the healthy, rests on evidence similar to that adduced in cases of typhus; and the same objections may be taken to the evidence which aims at establishing the spontaneous generation of the specific poison. There are causes, circumstances, or conditions which obviously favour the accession of relapsing fever, and no doubt, also, its occurrence in an epidemic form; and chief amongst these predisposing causes must be placed destitution and want of food, while the names applied to the disease by different countries indicate the popular belief as to such predisposing causes being credited with originating the disease in the first instance. Thus it is spoken of as the *famine fever* of the British Isles, and the *hunger pest* of Germany.

The Primary Paroxysm.—The seizure is generally, indeed almost always, sudden. Sometimes, on waking in the morning, or when employed in business, severe rigors at once come on, with a sense

of chilliness and frontal headache. These phenomena are more severe than their expression is in the commencement of typhus. There is slight prostration of strength from the first, but rarely so severe as in typhus. If premonitory symptoms exist, they usually manifest themselves by pains in the limbs and lassitude, nausea, and perhaps vomiting, with feeling of prostration. Subsequently, and very soon, febrile re-action sets in, sometimes violent, expressed by intense heat of skin, severe headache, throbbing temples, intolerance of light and sound, suffusion of face, sleeplessness, remarkable anxiety of countenance and jactitation, with a very rapid pulse—so rapid as to range from 110 sometimes as high as 140 beats in a minute; the tongue is coated with a white fur, and in a great majority of cases, in some epidemics, there is uncontrollable vomiting of greenish, bitter fluid, with or without epigastric tenderness, and great thirst. The pains in the muscles and joints are sometimes so severe as to resemble rheumatism, and when the pain in the back is severe, together with the rigors, the vomiting, and the headache, it may not be possible in the first instance to say that the attack may not prove to be one of small-pox. But the pain in the back is not generally so severe, nor is the vomiting so incessant in cases of *relapsing fever* as in cases of *small-pox*. The headache is to be distinguished from what is commonly called a "sick headache" or "bilious headache," by the circumstance that the "bilious headache" is in most cases *occipital*, and the heat of skin, combined with the quick pulse, serve to distinguish an attack of relapsing fever at its outset from one of "bilious headache." From idiopathic head affections the accession of relapsing fever is distinguished by the suddenness of the attack, the rigors, the hot skin, the pain in the joints and limbs, and the white tongue (DR. JENNER). The symptoms generally of *relapsing fever* are so severe that the patient takes alarm, and takes to bed at once. He does not feel weak, but he feels so giddy that he is unable to remain out of bed, or off the horizontal position. In some cases there is pleurodynia in a severe degree, but without any stethoscopic indications of pleural inflammation.

Pulse By the second or third day the pulse almost invariably exceeds 100; as a rule it reaches 120; in not a few cases it is as high as 140 or 160; and it is not rarely 140 on the second day of

the disease, being at the same time full, and of considerable firmness—symptoms not indicative of commensurate danger—with anxious and oppressed breathing. There may be also sweating, profuse and lasting for several hours, but without relief to the headache and other symptoms. Almost no sleep is obtained, and the little obtained is dreamy and unrefreshing. The skin continues dry after the sweating ceases; or after the primary rigors, if sweating has not taken place; and the heat of skin is ardent—as much as 102° to 107° Fahr.; and these febrile phenomena are occasionally varied by short rigors or slight sweating. Delirium does not generally supervene on the first attack, although, by the *fifth* or *sixth* day, just before the crisis, it has been in some cases of a violent kind. In a large proportion of cases there is decided jaundice, and in others the skin exhibits a bronzed hue. The jaundice is not attributable to any obstruction of the *ductus communis choledochus*, as bile passes freely, and even copiously, with the stools, and as, after death, the gall-duct is pervious. There is generally tenderness over the region of the liver in such cases; and it may be enlarged. Thirst is excessive; the appetite absent or voracious, and the bowels constipated. The tongue, at first moist, is covered with a white or yellow fur, which it may retain throughout the illness; and, in many cases, it may become dry all over, or with a brown dry streak down the centre, after the third or fourth day.

The Crisis.—After the patient has continued in this state for a period varying from five to eight days, a sudden change takes place, immediately preceded, in most cases, by an exacerbation of all the symptoms. "When every symptom appears hourly becoming graver—when the restlessness and general distress have reached their highest point—then ensues a most remarkable series of phenomena, followed by a remarkable intermission of all the symptoms, and an apparent restoration to health." This period has received the name of "Crisis," and supervenes generally on or about the *seventh* day, and its advent is rarely prolonged beyond the *eighth*. This change is ushered in by a most profuse perspiration, in some instances with an eruption of miliary vesicles, which breaks out from the whole surface of the skin, and in the course of a few hours the patient appears nearly well. More rarely the change is indicated by epistaxis as well as by perspiration, or by profuse

Top Durⁿ of Relapse 3 - 5 days

diarrhœa, catamenial discharge, or hæmorrhage from the bowels; and after either or all of these apparently critical changes have been established for a few hours, there is a complete and abrupt cessation of all the bad symptoms. The pulse quickly regains the natural standard, the tongue cleans, the appetite and sleep return, and the countenance resumes its tranquillity. This alteration is very often effected within a few hours, and on the following day the patient generally considers himself in all respects quite well, and may so continue to improve rapidly for *four* or *five* days. During this period, however, there are some patients who suffer from violent muscular pains in the limbs.

12-10¹¹
The Relapse or Recurrent Paroxysm.—About *seven* days after this critical change, or between about the *twelfth* to the *twentieth* day from the commencement of the illness, but generally on the *fourteenth* day, a sudden relapse occurs "in ninety-nine cases out of every hundred." This relapse commences suddenly, like the first seizure, by rigors, headache, loss of appetite, vomiting of green fluid, which is quickly followed by a hot skin, quick pulse, and a coated white tongue, confined bowels, followed by delirium, so that the phenomena may be exactly represented as a repetition of the first attack. In the interval of convalescence between the first and second attacks the pulse often becomes slow to an extreme degree, as slow even as forty-five to sixty beats in the minute; but, suddenly, on the relapse commencing, it again rises to 120 or more. In ordinary favourable cases perspiration would again occur in *two, three, four, or five* days, and the patient would be relieved as before. The chemical qualities of the sweat have never been determined in cases of relapsing fever; but it has a very sour and peculiar smell. In other cases, however, uncontrollable vomiting, great thirst, very rapid pulse, a hectic-looking circumscribed flush of countenance, jaundice, watchfulness, delirium, and death, may terminate the case.

WB
Termⁿ
in
Death

In some cases the relapse is very slightly marked, and indicated merely by a comparative increase in the rapidity of the pulse and a greater heat of skin than were present on the previous day. The duration of the relapse varies from a few hours to several days; the average being from *three* to *five* days, or less than that of the primary paroxysm. In some cases the relapse lasts less

In $\frac{1}{4}$ cases jaundice without obstruction present

OCCURRENCE OF SUDDEN DEATH IN RELAPSING FEVER. 471

than *twenty-four hours*; and in a few it is prolonged to *seven or eight days* (MURCHISON). The relapse is rarely prolonged beyond these periods in uncomplicated cases, but Dr. Lyons observed in the Crimea that the fever of the relapse was occasionally protracted to *twenty-one days* (Lyons *On Fever*).

If blood be taken from the arm, it is generally buffed, but it is *not* to be argued that *therefore* the lancet must be used in *relapsing fever*. So far as can be ascertained, no local inflammation attends it.

In nearly a fourth of the cases, according to Dr. Jenner, jaundice is present, and is sometimes intense. If present during the first attack, it may disappear before the relapse, and not recur; or it may occur only on the relapse: and it is important to notice, that while the jaundice continues, the stools still retain their natural hue, and may even be darker than common, and at the same time the urine may be frequently loaded with bile. Epigastric tenderness is most marked in the cases where vomiting occurs. When pregnant women are attacked with *relapsing fever* they usually abort, sometimes in the first paroxysm, but often in the relapse, and this event renders the prognosis more doubtful.

There is a tendency in *relapsing fever* to the occurrence of sudden death. It may happen by syncope, immediately after the critical periods, when the pulse becomes so very slow. It may also happen during the progress of the case, during either of the severe periods—namely, during the primary attack or during the relapse. It is indicated by a deep dusky hue of the face, lividity of the hands and feet, and a purple marbling of the whole surface. The trunk feels cool, and the hands feel cold, and without suffering any severe pain, or without sustaining any sudden discharge of fluids, a state of collapse insidiously comes on, from which the patient is unable to be roused, and death may follow in a few hours, generally from *twelve to twenty-four*, even after it was supposed that danger had been escaped. But death is a rare termination to relapsing fever; and when it does occur, the fatal event more commonly happens during the primary fever than during the relapse (DR. JENNER).

A second relapse, and a third, a fourth, and even a fifth, are reported to have occurred during epidemics of *relapsing fever*, but the cases are of rare occurrence.

Total duration fever 3 weeks

Convalescence

X

Duration of the Fever, and Convalescence.—Under ordinary circumstances, when there are but two paroxysms—*i.e.*, one primary paroxysm and one relapse—the total duration of the fever extends to about *three weeks*; and the convalescence is very slow—much slower than in typhus. The *relapsing fever* is very exhausting in its effects upon the constitution; and, dating the period of convalescence from the termination of the last attack, the time taken to recover is in most cases unusually long. To those, indeed, who suffer from more than one relapse, it is almost impossible to have health completely restored for a long time. They become a prey to various sequelæ of fever, or they continue sickly for months, with pallid countenances, puffed ankles, palpitations, extreme debility, noises in the ears, dimness of vision, diarrhoea, or dysentery. Dysuria is a frequent complication amongst women during the relapse. In many instances during the epidemic of 1847 and 1848 in Ireland, convulsions occurred in cases which otherwise seemed to be progressing favourably, and death invariably followed them. Dr. William Robertson observed in Edinburgh (and the Irish physicians record a similar observation) that delirium of a violent character occurred during convalescence, or after the critical discharge had taken place. It generally came on suddenly, with incessant talking, a rapid weak pulse, followed by perfect unconsciousness, flushed face, and contracted pupil.

WB

No special anatomical lesion has been pointed out as peculiar to *relapsing fever*. The most constant lesion is enlargement of the spleen, the size attained by that organ being on the whole larger than in either typhus or typhoid fevers. Dr. Jenner has recorded the weight in one case to have been as much as thirty-eight ounces, and of a size in proportion. Its substance is generally softened, sometimes diffluent. It is usually seen at its largest size when death occurs during the final paroxysm; but if death occurs during convalescence, the spleen is of a normal size. Occasionally pale, red, fibrinous infarctions are found in its substance and near its surface. They are easily broken down, have a fine granular fracture, and are considerably firmer than the surrounding tissue, from which they are separated by a distinct line of demarcation. As a rule, there is but little congestion of the lungs, the weights of which contrast singularly with the weights of organs in subjects dead of typhus fever.

Anatomical { Enlarged Spleen
 recumbent { Heart of any congestion of lungs
 + 2 { Thick blood etc.
 Increased no. of white cells

The blood in a few cases has been found fluid throughout the body after death; but generally, when drawn from the body during the febrile paroxysm, it is buffed; and decolorized coagula are found in the heart and large vessels after death more frequently than in cases of typhus. In several cases urea has been detected in the blood in considerable quantity. The proportion of white corpuscles is increased—a fact of interest in connection with enlargement of the spleen, and the state of anæmia so commonly observed (CORMACK, ALLEN, THOMSON, MURCHISON). The liver is generally large, and the gall-bladder filled with dark thick bile.

Sequelæ of Relapsing Fever.—One of the most common results is the occurrence of excessive pains in the limbs, more especially expressed about the knee and ankle joints; and even the long bones appear to be the seat of these pains in some cases. Combined with those local pains, the joints may swell; and the kidneys are in danger of being implicated. In some respects, therefore, the dangers are similar to those which attend scarlatina. The lymphatic glands are also liable to swell, and so is the parotid gland. Anasarca and furunculi may likewise supervene. 203

The most important of all the sequelæ, however, is a remarkable affection of the eyes—a form of ophthalmitis—which Dr. Mackenzie first described under the name of “*post-febrile ophthalmitis*.” It may occur during the course of the fever, but more often during convalescence, and even some months after convalescence has been established. It was very common in Glasgow after the epidemic of 1843; and assumed two different forms, namely,—(1.) An active inflammation of the shell of the eyeball and of the iris; (2.) An amaurotic state due to congestion of the choroid and the retina (DR. ANDREW ANDERSON).

These two forms of disease, Dr. Anderson observes, may be associated with two characteristics of the fever itself—namely, “the tendencies to visceral congestions, and to rheumatic-like pains;” while the constitutional character of the ophthalmia is in many cases proved by the unhealthy aspect of the blood, which flows dark, in some cases almost tarry, from the vein. Bleeding is found to be the most effectual—the only effectual—mode of cutting short this dangerous ophthalmia; and a very small loss of blood is found to be sufficient. This is especially noticeable,

Treatment Symptoms
to be combated

Vomiting
Headache
Anæmia
Jaundice
Murmur

because, during convalescence, tonics and quinine are most likely to be thought of.

Treatment.—All physicians agree that in the primary attack little medicine is required after opening the bowels by castor oil; or by five grains of the compound colocynth mass; or by two grains of blue-pill, and three grains of extract of hyoscyamus given at night, and followed in the morning by two drachms of the sulphate of magnesia in compound infusion of roses (MURCHISON). The symptoms are not readily under the control of remedies; the vomiting is often especially persistent. Five grains of calomel, with one grain of opium, has been found more efficient in subduing the severity of this symptom than counter-irritation or effervescing draughts. The violence of the headache in well-fed, or otherwise healthy patients, is best subdued by leeches or cupping; and in the poor, weakly, and ill-fed, by blisters to the nape of the neck, or by dry cupping there. Till the crisis comes, the symptoms may be mitigated, but not altogether relieved, and cases of ordinary severity are better left to nature, without interference on the part of the physician. Active purging is to be avoided; and the action of the kidneys is to be kept up by the frequent use of small doses of nitre (ROSS, HENDERSON, CORMACK, WARDELL, MURCHISON). "By keeping up the action of the kidneys from the first," Dr. Murchison justly entertains the hope that we may "prevent the occurrence of uræmic intoxication, which is one of the main causes of death in uncomplicated cases." He recommends the administration of the nitre as follows:—

From one to two drachms of nitre are to be dissolved in two pints of barley-water, acidulated with a drachm of dilute nitric acid, and sweetened with a little syrup. This quantity is to be used up during the twenty-four hours. Acetate of potash and nitric ether may be used for the same purpose; but the nitre has the additional advantage of keeping open the bowels.

The surface of the body should be frequently sponged over with cold or tepid water; stimulants are not usually necessary, but they may be required in the stage of languor or exhaustion ensuing on the crisis; or in cases where great debility has preceded the attack. If any anæmia exists, or if an anæmic murmur can be detected, stimulants must be given early. When jaundice

appears, Dr. Murchison recommends that nitro-hydrochloric acid should be given in combination with nitre, as in the following formula:—

Twenty minims of hydrochloric acid, with ten minims of nitric acid, every three hours, each dose diluted with the drink of nitre and barley-water already prescribed.

Contamination of the blood with urinary products is the great danger in cases of relapsing fever; and therefore, in all cases of relapsing fever, particular attention must be paid to the state of the urine, especially towards the period of the first crisis. When the daily amount is much reduced, or if entire suppression should ensue, and particularly if stupor, confusion of thought, or drowsiness should supervene, the bowels are to be freely moved by compound jalap powder, or by a turpentine enema. Determination to the skin should be promoted by the hot air bath; and saline diuretics may be given every two or three hours (MURCHISON). No means hitherto discovered will prevent the occurrence of the relapse. MB

FEBRICULA.

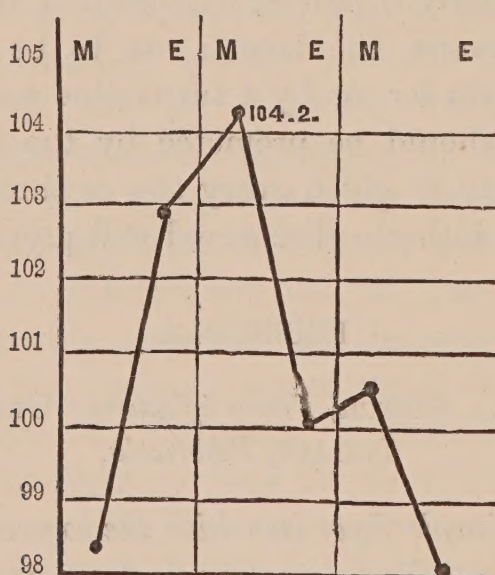
LATIN, *Febricula*; FRENCH, *Fièvre éphémère*; GERMAN, *Febricula*; ITALIAN, *Febbricola*.

Definition.—*A simple fever in which the expression of the febrile phenomena is of very short duration, lasting, as a rule, for twenty-four, thirty-six, forty-eight, or seventy-two hours or more, attended with a frequent, full, and often firm pulse, white and coated tongue, pains in the loins and limbs, thirst, constipation, a scanty discharge of high-coloured urine, hot and dry skin, sometimes an eruption of roseola or erythema about the loins or thighs, coming and disappearing with the fever (MOREHEAD); severe headache, sometimes acute delirium, and flushed face. The subsidence of the fever is generally associated with copious perspirations, or herpetic eruptions.*

Pathology.—We do not know of any specific poison as the cause of such phenomena as those detailed in the definition; neither have we any evidence that febricula is a contagious or miasmatic disease. There are many different causes which are known to be capable of exciting expressions of febrile phenomena similar to those mentioned in the definition,—such as exposure to

great heat or cold, surfeit, inebriety, mental or bodily fatigue or excitement; and specific poisons, in uncertain or otherwise mild doses, such as the typhus or enteric fever poisons. It is also associated with local and functional disturbances—*e. g.*, catarrhs (bronchial, gastric, intestinal, urethral), milk fever, the fever of alcoholism. Such cases are especially characterized by the apparent severity of the febrile state, the shortness of its course, and the absence of any local complication or specific eruption.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF FEBRICULA. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).

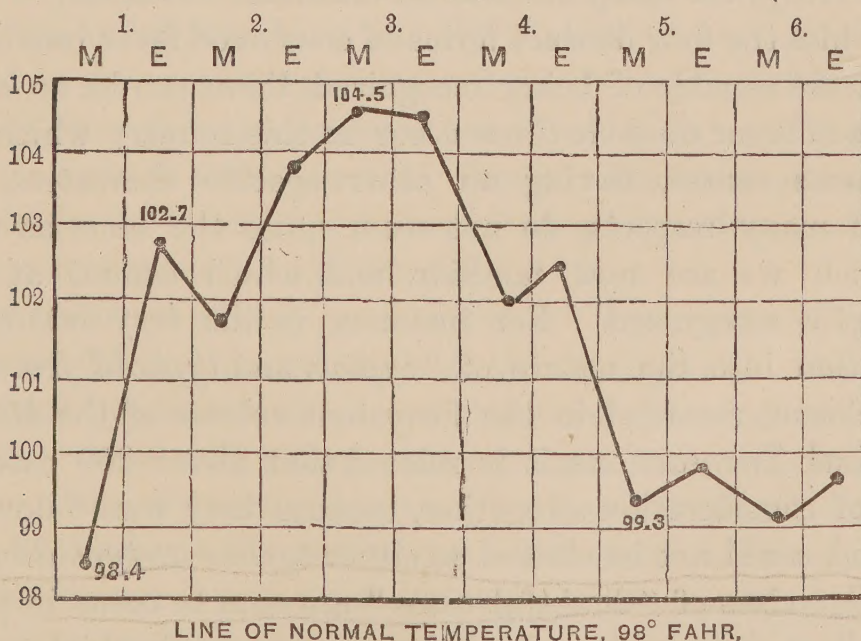


LINE OF NORMAL TEMPERATURE, 98° FAHR.

The pathology of such apparently simple fevers demands extensive investigation, and especially in the tropics, where febricula is a very common disease. The *ardent fever*, the *sun fever*, the *common continued fever* of Burmah and India generally, are all names which indicate severe or protracted cases of febricula—cases of fever which “differ in degree rather than in character” (MOREHEAD). They are common in those parts of India which do not experience much of the influence of the monsoon rains. Cases of true febricula commence with chills, followed by re-action, and this by perspiration. They are characterized by a quick and comparatively sudden rise of temperature, as indicated by the preceding diagram—a rise of temperature in a few hours to 4°, or 5°, or 7° above the normal temperature of 98° Fahr. They have thus a sudden beginning

and a rapid arrival at a maximum—phenomena which are only shared in by some forms of *malarious fever* (*intermittent*), *variola*, *measles*, and *pneumonia*. The defervescence also is characteristic. In febricula the maximum of temperature may only last for a few hours, or a single day, when the defervescence sets in rapidly; so that in twenty-four or thirty-six hours the body will have returned to its normal heat—an example of *pure crisis*. No other febrile disease gives expression to similar phenomena; and the co-relation of temperature to the other phenomena, especially to the excretion of urine, is also peculiar. The urine presents, during this disease, the very type of febrile ~~X~~ urine. On the second or third day, according to Dr. Parkes's own observations, the amount of urine is extremely small (twelve to twenty ounces), of very high specific gravity (1035-1037), with the solids and sulphuric acid very much over the average, and the amount of urea large. When the temperature falls, the quantity of urine rapidly augments. The increase of urea and of the solids is not so much, however, as in the height of the more severe and prolonged fevers (Parkes *On the Urine*, p. 243).

TYPICAL RANGE OF TEMPERATURE IN A CASE OF PROTRACTED FEBRICULA (*ephemera protracta*). THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).



In cases of more protracted febricula, the early phenomena are similar to the shorter cases, and the protraction is mainly due to the slowness of the defervescence—an example of *lysis*.

The phenomena of such defervescence, as indicated by the range of temperature, are shown in the foregoing diagram.

As a rule, these fevers are not serious; but the degree of re-action has always a relation to the state of the constitution, whether sthenic or not (MOREHEAD).

Treatment of Febricula.—Such means as emetics, purgatives, tepid sponging, diaphoretics, and antiphlogistic regimen are to be employed. In plethoric individuals, where there is much headache and flushing of the face, a moderate general blood-letting, or leeches to the temples, may be expedient, but such remedies are not often necessary (MOREHEAD).

SIMPLE CONTINUED FEVER.

LATIN, *Febris continua simplex*; FRENCH, *Fièvre continue*; GERMAN, ———; ITALIAN, *Febbre continua semplice*.

There are not a few physicians who doubt the occurrence of *simple continued fever* as distinct from the continued fevers already described. On the other hand, there can be no doubt that cases of an anomalous or mixed nature do sometimes occur, concerning which a decided diagnosis cannot be given from the general symptoms merely; and the term *continued fever* “has become a refuge for many cases of an uncertain character.”

Thus while the four distinct forms of continued fever previously described are capable of being recognized, there can be no doubt that cases of fever do sometimes occur in this country which run a continuous course, having no other specific characters, and which, in many respects, do not seem quite the same as those with which we are now familiar, and which cannot at once be clinically recognized. For instance, in the very interesting investigation into the nature of “*typhus* and *typhoid fever*,” by Dr. Murchison, recorded in the forty-first volume of the *Medico-Chirurgical Transactions*, it is related that about 200 cases are left out of consideration altogether, because they were “doubtful cases,” and could not be classed as either *typhus* or *typhoid* cases.

A similar class of “doubtful cases” are seen to occur in places where *yellow fever* and *remittent* fevers occur, and which cannot be classed as either the one or the other form of fever.

Again, in the Mediterranean latitudes, there is a “gastric remittent” fever described, which seems to have many char-

acters in common with some of the forms of continued fever (CRAIGIE, MARSTON).

Wunderlich and Murchison both describe febrile phenomena which are of so anomalous a kind that they refer them to a combination of the poisons of typhus and typhoid fevers, so that the characters of each do not remain distinctive. So likewise Dr. W. T. Gairdner, in stating that of late (from 1853 till 1862) the cases of fever in the Edinburgh Royal Infirmary have not been more than seven or eight cases a month under his notice—including numerous anomalous fevers which have prevailed, and which have sometimes quite overborne the numbers of genuine *typhus* and of *enteric* fever together (*Clinical Medicine*, p. 154). A fever termed "*gastric*" is distinctly described by Dr. Andrew Anderson, of Glasgow, which would also come under this head. The "*bilious remittent*" or "*bilious typhoid*," of Greisinger, of Tubingen, is another form of continued fever which requires investigation. He observed it at Damietta, in Egypt. It is probably a malarious fever of a remittent type, or some form of yellow fever. The sudden fall of the pulse from 120 to 75 was not attended by corresponding improvement of the patient, but was the forerunner of severe typhoid symptoms and jaundice. The mortality was equal to 19 per cent.; and quinine was found to be of signal service (MURCHISON).

The works of Morehead and Sir Ranald Martin may also be referred to for various anomalous forms of continued fever; and in which the "ardent continued fever" of India may be quoted as an example of a very serious disease. For an account of this fever the reader is referred to page 164 of Dr. Morehead's work, and page 204 of Sir Ranald Martin's.

It is these "doubtful or anomalous" cases especially which require careful and special methods of investigation. They are of the utmost importance to science, for more extended information regarding them will either connect them with forms of fever between which they seem to stand; or these "doubtful cases" will eventually separate themselves into distinct forms, whose history is still unknown. In such doubtful cases observations regarding the co-relation of temperature, the excretions, the succession of phenomena, and general course of the disease, are imperatively demanded.

The poisons of tropical fevers especially require to be carefully

studied, and the phenomena of the febrile state which accompanies them embrace some medical problems of the most abstruse nature. Physiological data of an exact kind are now beginning to rise around us, which will give a standpoint for comparison in the study of the phenomena of fever in the tropics. Extremely important observations are being worked out by Dr. Emile Becher, of the Army Medical Department, regarding the influence of tropical climate on the excretions of the urine in relation to the body weight. At great personal sacrifice and denial of self, he has twice undertaken such investigations on his own person in voyages to India, round the Cape of Good Hope. With all such exact information, and the improved physical aids to investigation, it behoves the physician and pathologist to investigate medical problems with the same logical rigour and severity as a chemical or an astronomical theorem demands. On this important point the opinion of one may be especially quoted, whose experience as a teacher of clinical medicine has been great, and whose philosophical investigations into the nature of fever, in particular, command the respect of all. On this point Dr. Parkes thus writes,—“The power of observation in medicine is a kind of tact which ought to be cultivated with the same assiduity as the chemist practises when he learns how to manage his delicate manipulations, or the astronomer when he wields his wondrous tube. In medicine the observation and recording of phenomena has been held to be an easy and trifling task, which any tyro was competent to do. Hence half the error and uncertainty of medicine. Inaccurate, that is, erroneous and incomplete observation, has been the cause that, till within these few years, the fevers of cold countries have been so absolutely uncomprehended, and that the fevers of hot countries are still shrouded in obscurity. The most valuable addition any one could at present make to our knowledge of tropical fevers would be a simple record of all the cases in an epidemic. These cases should be observed with the keen tact of a Chomel, and recorded with the fidelity of a Louis. We want no explanation or word of comment added to them; we want merely the cases. Then, when the numbers are sufficient, we should certainly begin to put order into this chaos. And let not any one who may have the opportunities be deterred from the task by that fallacious and, we beg to say, most reprehensible argument, with which some people may favour him,—viz., that

his cases will be 'tedious,' 'heavy,' and 'unread.' Unread they will be, certainly, by some of the profession, who consider their routine practice as great an effort as their intellect will bear; but read and analyzed, we will venture to say, they will be by those who think no labour too great if they can fix safely the foundations of medicine, and for whom, if *accurately* reported, no cases can be too long, no observations too minute. Only, before the task is commenced, let the observer feel that his powers are equal to it; and let him bear in mind the example of Louis, who recorded most carefully for a long time, that he might train himself to this duty, and then, throwing his probationary cases aside, as too uncertain for use, began to make those remarkable series of observations which have linked his name for ever with the greatest improvements in modern medicine—the employment of a correct method of studying his science" (*Brit. and For. Med.-Chir. Review*, Oct., 1850, p. 435).

[SPECIFIC] YELLOW FEVER.

LATIN, *Febris flava*; FRENCH, *Fièvre jaune*; GERMAN, *Gelbes Fieber*—Syn., *Gelbfieber*; ITALIAN, *Febbre gialla*.

Definition.—A specific malignant fever of a continuous type, occurring, as a rule, only once during life, and propagated by contagion. It is attended by yellowness of the conjunctivæ and skin, delirium, suppression of urine, interstitial hæmorrhages, and hæmorrhages from the stomach, mouth, nares, and rectum (black vomit, black stools), a slow and, at times, an intermittent pulse. It is limited to very definite geographical limits, never having been known to propagate beyond 48° north latitude, nor without a temperature of 72° Fahr. at least. It has been imported into Lisbon, into St. Nazaire, in the department of the Lower Loire, into Plymouth and Southampton, where cases have run their course and proved fatal. It has been imported and become epidemic as far south as Monte Video. It has occurred as high as 4,000 feet above the sea-level (Newcastle, in Jamaica). But, as a rule, it is endemic in low districts on the sea coast, and rarely occurs above an elevation of 2,500 feet above the level of the sea.

Pathology and Symptoms.—Yellow fever of a specific kind must now be regarded, from an enlightened consideration of its history, as one *sui generis*, and specifically different from

the remittent and intermittent fevers, in which the patient may become yellow, or any other form of malarious yellow fever (CULLEN, CHISHOLM, BLANE, WOOD). There are those who believe that these fevers are the same in kind, but various in degree; that certain atmospheric conditions, such as great heat or humidity, acting on a predisposed frame, will produce all the symptoms of the most malignant fever; that the intensest form of yellow fever is but the developed degree of the common bilious derangements peculiar to hot and rainy seasons (TOMMASINI, CLEGHORN, LIND, HUNTER, ALISON, CRAIGIE, MARTIN). It has been held also that a specific non-contagious agent produces a fever which has been called "yellow," but which is totally different from the real yellow fever (ROCHOUX). On looking carefully into the history of yellow fever, on which volumes have been written, the conclusion arrived at seems to be,—(1.) That there is a specific yellow fever, propagated by a contagious virus or poison which multiplies itself by its passage through the human system, and which reproduces the same specific true yellow fever. The type of this fever is continuous. Pyrexia, delirium, suppression of urine, black vomit, are the leading symptoms of this fever—the *hæmagastric pestilence*, as it has been also called. (2.) That there are other fevers, and especially severe marsh fevers, in certain geographical limits, which have a close resemblance in symptoms to the contagious and specific yellow fever. So also, it is said, have fevers arising simply from a high temperature acting on an unseasoned subject. On this point my friend and colleague, Dr. Maclean, Professor of Military Medicine, who has had twenty-two years' experience of East Indian fevers, writes me as follows:—

"I am now myself a firm convert to the doctrine that yellow fever is specifically distinct from remittent. To this opinion I have come with a full knowledge of the fact that some cases of remittent fever in India closely resemble some of the forms of yellow fever. But of this I am now certain, that the yellow fever of the true yellow fever zone is unknown in India where true malarial fevers abound. There is in true yellow fever, for the most part, an absence of that periodicity which is an unfailing characteristic of true malarial fevers. Then there is the difference so well insisted upon by Blair in true malarial fevers. Men do not pass from recovery to health, as is the case in such a marked degree in yellow fever, after which there is no, or very little, evidence of the existence of any cachexy. Malarial fevers exist and are destructive at a

temperature at which yellow fever is at once destroyed. Albuminous urine is almost invariable in yellow fever—only occasional in remittent. There is in yellow fever an unexampled range of hæmorrhages; in remittent fever these hæmorrhages are often, indeed generally, absent. Quinine has a power over malarial fevers that is beyond the reach of doubt or cavil; the same is not true of yellow fever. Men suffer from malarial fevers again and again; second attacks of yellow fever are, to say the least, rare.”

The correctness of the above view of this important question has also received a remarkable illustration in some observations made in Mexico by the Medical Staff of the French Army serving there.

Yellow fever had disappeared from Vera Cruz. In the month of October, in the middle of a sudden augmentation of sickness, several severe cases of a disease like *vomito* appeared. The physicians dreaded a return of yellow fever in its epidemic form, in spite of the relative abatement of temperature and the almost constant prevalence of a north-east wind. These fears were augmented by the disembarkation at this time of a great many fresh troops; also by the suddenness of the invasion of the disease, showing itself in a great many cases by hepatic symptoms, bilious vomiting, fever, prostration, articular pains; the fever being continuous, or at least without sensible remissions, in many cases. But soon many circumstances demonstrated the groundlessness of the fear, and showed that the disease was malarious. These were,—(1.) The quickly recognized efficacy of quinine, which in the month of May, when true yellow fever raged, gave only negative results at the best; (2.) The rapid supervention of splenic enlargement, often attended with pains; (3.) The small relative mortality; (4.) The absence of hæmorrhages, other than occasional epistaxis; (5.) The constant absence of albumen in the urine; (6.) The difference in the anatomical lesions, and chiefly in the presence of enormously enlarged and softened spleens; (7.) The absence of all the lesions characteristic of *vomito*, such as the yellow colour of the body, with marbled marks, black matters in the intestines, discolouring of the liver approaching to yellowness; (8.) Finally, the non-immunity of soldiers of the garrison who had already suffered from yellow fever (*Recueil de Memoires de Medecine, de Chirurgie, et de Pharmacie Militaires*, No. 37, Janvier, 1863).

Considering true yellow fever, therefore, as one of the specific continued fevers, having a certain limited geographical range, it

is necessary at the outset to define what is meant by true or pestilential yellow fever, and what are the diagnostic symptoms which distinguish it from the diseases which resemble it, but which are really dissimilar.

It may be asserted unconditionally at the outset, that the significance of the symptoms of yellowness of the skin and black vomit is very small indeed as diagnostic marks. Different shades of yellowness of the skin have been described as forming a prominent symptom, not only in epidemics of yellow fever, but by all writers on the fevers generally of hot countries. Yellowness of the skin in remittent fevers arising from malaria has been noticed in all climates, although it is certainly most common in those of the western hemisphere. Cleghorn observed it at Minorca; Irvine in Sicily, in the autumnal fevers; Burnett in the Mediterranean fevers, of all depths of colours. In the fatal fever of the Mysore country, yellowness during some years has been almost universal; so also in Batavia, and in the fevers of Rangoon in 1824-25. A fever attended with yellowness of the skin raged like a pestilence in Rohilcund from 1836 till 1840, at the same time that a fever with symptoms of plague was prevalent in Marwar and Meywar, and common remittents and intermittents prevailed between these districts. A fatal remittent fever attacked Her Majesty's 29th Regiment in 1844 at Ghazepore. In many cases there was "deep jaundice;" and in one case a symptom occurred which has been often witnessed in the West Indies—namely, sloughing of the penis and scrotum (Parkes "On the Contagion of Yellow Fever," in *Brit. and For. Med.-Chir. Review*, Jan., 1848). So also in some forms of specific yellow fever, as in the *algid* form, so well described by Dr. Lyons, in the Lisbon epidemic of 1857, yellowness was very often wanting, "many cases dying without having ever exhibited a trace of yellowness on any part of the cutaneous surface, or even the conjunctivæ during life" (Lyons *On Fever*, p. 338).

Black vomit is an event which occurs in fevers of marshy origin, and in the so-called "seasoning fevers," as well as in gastric affections of a purely tropical nature, in *coup de soleil*, and in some injuries of the brain. In the remittent fevers of the African stations, black vomit is not an unusual occurrence. So also in some of the yellow fevers of America, which are of marshy origin, black vomit is a usual symptom (BOOTT).

These two events—namely, yellowness of the skin and black

vomit—being of themselves insufficient as diagnostic marks of true or specific yellow fever, additional grounds of difference are found,—(1.) In the type of the fever, which is continuous and not remittent; (2.) In the fact that it occurs, as a rule, only once during life; (3.) In the fact that it is propagated by specific media from infected persons or places to others. But although in no one of those phenomena, taken singly, except in that of its communicability from person to person, do we find any definite characters to rely upon to prove the existence of a formal and specific yellow fever, yet, in the general assemblage and collocation of symptoms, peculiarities do present themselves which are easily discernible by an experienced eye.

Though the subject of tropical fevers is too little known to warrant decided opinions on many points, yet the true yellow fever, or hæmagastric pestilence, is now so clearly stamped with characters so peculiarly its own, that it takes its place as a specific fever of a continuous and generally rapidly fatal type. Its pathology is best exemplified in the history of such isolated outbreaks of it as are to be found in the cases of the "Hussar" (BLANE), the "Bann," the "Kent," the "Scout," the "Eclair," the "Hankey," the "Icarus," the Lisbon epidemic of 1857, and the importation of the disease from Havanna into the port of St. Nazaire by the "Anne Marie" in 1861; and no description of yellow fever can be complete which does not give an account of some of these remarkable instances of this disease.

An analysis of all the circumstances connected with the "Eclair" shows (1.) That the immediate consequences of landing the crew at Boà Vista were a thorough intercourse with the inhabitants, and the communication to them of the same fever with which the "Eclair" was infested. For some time before the arrival of the "Eclair" it is certain that the island of Boà Vista was perfectly healthy; and this was true also of all the other islands of the group (ALMEIDA, MACWILLIAM). So great, also, was the dread of the disease among the inhabitants that the consul had great difficulty in procuring labourers; nevertheless, the crew managed to smuggle vast quantities of spirits, and, of course, it is possible that more secret intercourse went on than can be gathered from any official reports. Certain of the inhabitants were also brought more or less in contact with the crew of the "Eclair." There were—1st. The military guard at the fort; 2d. The labourers

employed on board the "Eclair"—forty-one in number; 3d. The labourers employed in the launches, or at a coal-heap on a small island—forty-six in number; 4th. Washerwomen who washed the officers' clothes—seventeen in number. In addition to these, Captain Estcourt, the commander of the steamer, lived at the consul's house; the gun-room and ward-room officers and midshipmen occupied a house in Porto Sal Rey; and leave was given to the warrant officers and a few of the men, one of whom stopped in the town for two nights. The "Eclair" left on the 13th of September; on the 14th or 17th a corporal of the guard was taken ill; on the 15th or 18th, a private; and in both cases the symptoms were fever, wildness, and constant black vomiting. The corporal died on the 17th or 20th, and the private on the following day. Others of the guard were taken ill, and, being conveyed into the town, the introduction of the fever amongst the inhabitants is attributed to them. It also appears (2.) That the men who were chiefly in contact with the crew and with the sick men, and who were in the sick men's apartments, suffered much more severely than any other class; (3.) That the propagation of the fever appears to have been strictly in proportion to the amount of intercourse; (4.) That within a reasonable time after the departure of the "Eclair" there were three persons ill with fever at Boà Vista, and two already dead—all of whom had been in contact with the crew of the steamer.

The period of incubation of the fever at Boà Vista was found to vary from two to eight days; and the facts recorded in the history of the spread of the fever over the island show that certain persons living nearest and most in contact with the two sick soldiers were first attacked. When the disease appeared with great virulence in the island of Grenada in 1793, its spread by infection first attracted notice by the arrival of the "Hankey" from Bulam, on the west coast of Africa, on the 19th February, 1793, some days before the fever broke out on the island. In this vessel, at Bulam, the fever had prevailed for five months before, to a great and fatal extent (CHISHOLM, SIR WM. PYM).

A most interesting and consistent account of a yellow fever outbreak has been given by Surgeon J. D. Macdonald, Esq., F.R.S., in the medical journal of the "Icarus" (*Annals of Military and Naval Surgery*, p. 126). Mr. Macdonald's account is especially interesting in the following particulars:—(1.) It tends to fix

more definitely than has yet been done a period of incubation. (2.) It shows that in certain localities specific yellow fever is always in existence, expressing itself by sporadic cases every now and then, especially in the West India Islands, as a central focus to that geographical district where the disease is endemic, and where it has assumed in some places an epidemic character. (3.) It tends to corroborate the circumstantial evidence already accumulating, that the disease may be propagated by *fomites*, much as in the case with typhus and scarlet fevers. (4.) As with typhus and typhoid fevers, so with yellow fever, "the doctrine of its spontaneous origin" can have no foundation to satisfy the rational mind.

Bearing on the *period of latency*, Mr. Macdonald shows that yellow fever was rife at St. Domingo when the "Icarus" arrived there, and the professional zeal of Dr. Maclagan, the assistant surgeon of that ship, led him to visit some half-dozen cases on shore at Port-au-Prince. He is believed to have visited these cases on the 10th of June, and the first appearance of the symptoms which ended in his much-lamented death betrayed themselves on the 24th of June—i. e., *fourteen* days after having first visited the sick, and after the ship had been at sea four days. Fourteen days is not uncommon as the period of latency for the variolous poison. Another case is cited—that of a boy, Lambert—which shows that the period of latency could not have been less than *eight* days. The experience of the Lisbon epidemic marks the time of incubation as varying from two to ten days, and in some instances extending to fifteen days. The importation of yellow fever by the ship "Anne Marie" into St. Nazaire—a town in the department of the Lower Loire, about 47°30' north latitude—in the summer of 1861 (*Ann. d'Hygiene*, Oct., 1863, p. 416), confirms the belief in a lengthened period of incubation; and from a careful analysis of the history of specific yellow fever cases, I think it will be found that the period of incubation tends to lengthen with the transportation and propagation of the disease into latitudes the most remote from the equator.

The history of the importation of the disease into St. Nazaire is as follows:—About the 13th of June the "Anne Marie," a wooden sailing vessel, laden with cases of sugar, left Havanna, having been there a month during the prevalence of a severe epidemic of yellow fever. None of the sailors suffered so long

as she lay at Havanna, except from a little depression, loss of appetite, and a certain tendency to vomiting. After leaving Havanna for France there was no sickness for seventeen days. On the 1st of July two sailors were attacked (without precursory symptoms) with violent shivering, pallor of the face, injection of the eyes, congested lips, and continued delirium. One died in twenty-three hours, the other in one hundred and ten. On the following seven days other persons were attacked, making in all nine cases out of a crew of sixteen persons. Only two deaths occurred. The ship arrived at St. Nazaire on the 25th of July with seven men still sick, but all of them convalescent, thirteen days having elapsed from the date of attack of the case last taken ill. Therefore, having had no deaths and no fresh cases for ten days, the "Anne Marie" was not placed in quarantine at the end of her voyage on arrival at St. Nazaire. Near her, as she lay in that port, there were anchored two ships of the imperial navy—namely, "Le Chastan" and "Le Cormorant," the former touching her. Three other ships, "L'Orient," "Les Dardanelles," and "L'Arequipa," were also near her. According to the custom of the port, the sailors of the "Anne Marie," being only engaged for the voyage, quitted the vessel on her arrival, and were dispersed throughout the town. The commander, who had been ill, went home, the vessel was left to the second in command, and the places of the men were taken by seventeen fresh men, to discharge the cargo of sugar. These men were strong, very robust, and completed the discharge of the ship in eight days. Of these men twelve or thirteen were attacked with yellow fever, and many of them died.

"Le Chastan," having been close alongside the "Anne Marie," left on the 29th July, and sailed to Indret, on the Loire, 44 kilometres distant. Her crew, five in number, seemed in perfect health when they arrived at Indret; but on the 1st of August (*i. e.*, three days after leaving St. Nazaire) a man fell sick of yellow fever; then the remaining four by the 5th of August were all ill; and by the 10th of August all the five men, the crew of "Le Chastan," were dead. These men had all been on board the "Anne Marie" for about a quarter of an hour. "Le Cormorant" quitted St. Nazaire on the 10th: her crew, six in number, were then in perfect health. On the 14th of August two of her men were taken ill, and died on the 26th, of decided yellow fever.

Eight vessels in all had been near the "Anne Marie," and sick patients had gone on shore at St. Nazaire and its neighbourhood, communicating yellow fever to two or three, and slighter illness of the same kind to other persons who were about them. Altogether, it is on record that forty-four cases of yellow fever were communicated by the "Anne Marie," resulting in twenty-six deaths. During this period the heat was more like that of a tropical than of a European climate; but neither yellow fever nor anything like it had ever been seen in the district. It cannot now, therefore, be disputed that, by the arrival of the "Anne Marie," yellow fever was imported into St. Nazaire, and by propagating itself occasioned an outbreak of yellow fever at that port. And when it is remembered that fatal cases of specific yellow fever have already occurred at our own doors,—in Plymouth Sound and Southampton Water,—there is a like possibility of the importation of yellow fever into English ports under climatic conditions favourable to its development, and when we might least expect it, through the rapid and frequent communication which we now enjoy with the West India Islands, the very central focus of yellow fever. "We have no more reason to trust in our own fancied security than the people of Monte Video had before yellow fever made its appearance amongst them" (J. D. MACDONALD). The *Lancet*, of Feb. 12, 1863, contains "An Account of Yellow Fever as it occurred on board the Royal Mail Steam-ship 'La Plata,' in the month of November, 1852," from the pen of my friend Dr. Wiblin, the medical superintendent of quarantine at the port of Southampton. He there shows that fourteen cases of yellow fever had occurred on board the "La Plata" during her homeward voyage to Southampton from the West Indies. On the morning of the 18th November she arrived at Southampton, where she was kept for two days in quarantine. On the morning of the 28th the fourth engineer of the "La Plata" (Mr. Napier), who had been lodging in the town for eight days, was seized with symptoms of yellow fever, and, after a week's illness, died of the disease. No other cases occurred in the town. The "La Plata" was a wooden ship; and Mr. Napier had been at work on board of her during the time when he was lodging in Southampton.

In connection with the extension or propagation of specific yellow fever beyond its usual geographical limits of constant existence, "it is a remarkable fact," says Mr. Macdonald, "that

the deaths from yellow fever at St. Thomas's, before it became a coaling depôt for the Royal Mail steamers, as compared with the deaths afterwards, bear the proportion of 4 to 64. And in connection with this, must also be mentioned an equally important fact, that the combustible and other qualities of coal, exposed to the weather as it is at that island, became much deteriorated." How far this loss may prove to be a *pabulum* to yellow fever poison is unknown, but the covering in, as well as the enclosure of coal in tropical climates, as Mr. Macdonald notices, is worthy of the attention of the proper authorities.

All the cases, like the "Eclair," the "Bann," the "Imaum," the "Icarus," the "Anne Marie," the "La Plata," agreeing as they do in all their main features, it is impossible to doubt the existence of a multiplying infecting virus as the specific cause of yellow fever; and in cases where it was transported, imported, and propagated, the fever, except in certain cases, seems to have arisen only in persons who had been exposed to whatever deleterious influence was exercised by the atmosphere of ships in which cases of yellow fever existed. Moreover, the facts connected with the "Imaum," the "Icarus," and the "Barracouta," show that infected places and persons are alike dangerous to those who are at all susceptible; and in the cases of the "Imaum" and the "Anne Marie," it was shown that a disease taken in a certain locality, and spreading from person to person, may finally affect a second locality through their medium. It appears also to be quite a mistake to suppose, as Mr. Macdonald observes, that no individual can communicate the disease to another unless he himself is actually under its influence at the time; or, secondly, to consider such an individual as differing in any essential particular from an infected locality. Indeed, a ship itself is only an individual on a grander scale. This is the view Mr. Macdonald takes with regard to the "Imaum" receiving the disease from the officers of the "Icarus;" and it is also the view which explains the events which followed the contiguity of the ships to the "Anne Marie" in the harbour of St. Nazaire. To some extent the disease spread through personal intercourse to persons who were not near the ship,—in one very important case, that of M. Chaillon, a physician at Indret, who is said never to have been near the ship nor the town of St. Nazaire, but who contracted infection from four labourers who came

infected from the ship, and whom he attended medically at their houses. He contracted yellow fever, and died. In a second case, one of the ship labourers, who himself had yellow fever, is said to have carried the infection certainly to his wife, and perhaps to an old man in whose house he and his wife lodged. All of them were attacked with yellow fever—the old man fatally. Unquestionably with regard to the “Anne Marie,” and doubtless also with regard to the other ships, *the ships themselves*, irrespectively of sick persons in them, were *foci* of yellow fever infection. The men therefore, no doubt, carried infection passively, as they might have carried an odour from the ship, or as a student carries the smell of the dissecting-room on his clothes, especially felted textures. “I have often looked upon my own monkey-jacket with horror,” writes Mr. Macdonald, “as the possible means of communicating so formidable a disease to others.” Men thus labouring in the hold of infected ships, without themselves contracting yellow fever there, might carry infection to their homes, in climates and places where yellow fever may prevail. And it is a question how far, like cholera and typhoid fever *excreta*, the poison of yellow fever may not increase in places where decomposing animal material abounds.

With regard to the Lisbon epidemic, it has been proved that the epidemy remained concentrated in Lisbon, and did not extend to its neighbourhood, nor to any other part of the kingdom, although the communication by land with all the towns and villages continued active and uninterrupted. In many places there appeared cases of yellow fever, evidently brought from the capital, but in no place was the disease transmitted or propagated in an epidemic form. The official report records, that of 182 persons who left Lisbon for different places in Portugal, carrying with them the germ of yellow fever, which broke out or developed itself in them after their arrival at those places, 86 of them died. *In no instance was yellow fever communicated from them to any other person in the places whither they went.*

As to the origin of the disease in Lisbon, it appears certain that the first cases were amongst men employed in the Custom House, or with persons in close communication with them. It showed itself in the streets where these people lived. The disease, which commenced in July, did not become epidemic till September; but it remained stationary for awhile in the parishes where it first

broke out, and then spread gradually and regularly to other parts. It is therefore presumed that the disease did not proceed from any general cause operating on the whole mass of the population, such as would come from meteorological or hygienic conditions. It was not persons of different classes, and living in different parts of the city, who were attacked at the same time; on the contrary, it commenced with one distinct class, between the individuals of which there was much communication, and went from house to house, from street to street, without invading more than a certain extent of the city. It is presumed, but not proven, that the disease was imported through the Custom House, where luggage and effects are opened to be passed. Many of the ships which arrived at Lisbon during the epidemic and in the previous months had cases of yellow fever and losses during the voyage; but the only case proved was in the "Tamar," which arrived in March from Brazil, the captain of which owned that he had two deaths from yellow fever on board during the passage. This ship arrived again in September, and reported at Southampton two cases which she had had on board. Lisbon and Oporto have been very subject to these importations since 1849, when yellow fever became epidemic in Brazil. In the report it is concluded,—(1.) That Lisbon is not to be considered as liable to *spontaneous* outbreaks of yellow fever. (2.) But, from its latitude and local conditions (especially inasmuch as it abounds in filth in some localities where population is dense and sewerage imperfect), it is favourable for the development and spread of specific yellow fever. (3.) It is concluded that the epidemic of 1857 was imported from Brazil.

Symptoms.—Uniformity in the order and character of the symptoms of specific yellow fever must not be looked for. All the best writers on the subject, whether recording their experience in the West Indies, the West coast of Africa, or the south coast of Spain, are unanimous to the contrary, and consequently, while direct and faithful descriptions may have been given of each epidemic, yet the results are not general nor uniform. Certain symptoms in certain epidemics vary in their nature and in the time of their accession, while others common to former visitations are wanting in those which follow. Sometimes the full complement of standard symptoms are present, sometimes they are imperfect and deficient, and sometimes displaced. At one time the

diagnostic symptom in an epidemic is the supra-orbital headache. At other times the tongue symptoms are alone diagnostic, or their equivalents are expressed in the uvula and fauces. Intense surface heat, albumen early in the urine, and early black vomit, are among the later symptoms; and smoky pale urine, with perfect blood-corpuscles, take the place of the straw-coloured or bilious urine, with its sediment of tube-casts and epithelial matter.

The general appearance of the tongue in well-marked cases is redness of the tip and edges, with prominent papillæ and a creamy surface. Subsequently the fur separates from its middle surface, and lies in white wavy flakes; and the next series of changes consists in separation of the epithelium, which begins at the tip, proceeds to the edges and down the raphe, and may continue till the whole surface is denuded, the papillæ obliterated, and the tongue becomes smooth and dryish, of the colour and appearance of raw beef. Such a condition is generally associated with exudation of blood so free as to coat the mouth and tongue, and collect upon the teeth. The tongue is then usually much smaller and more pointed than in the earlier stages.

A rare manifestation of capillary irritation in yellow fever consists in an efflorescence of the skin in the form of a subcutaneous rash on the chest, and extending over the abdomen and arms. Rose-coloured spots of a somewhat circular shape have been noticed on fine, delicate, sensitive skins, varying from the size of a flea-bite to what might be covered with the point of the finger. They result generally from mosquito wounds, and become hæmorrhagic at the end of the disease, when it terminates fatally.

Bloody furuncles appear late in the order of symptoms, and are to be regarded rather as sequelæ. Their most common site is on the wrist, over the metacarpal joints, along the front of the legs, below the scapulæ, and over the hip, in the parotid region, and over the forehead and lip. They are generally in close proximity to the smaller arterial branches, such as the ulnar, radial, anterior, tibial, gluteal, intercostal, and facial arteries. They become tender, acuminated, and inflamed, and sometimes form large abscesses of purulent matter, with a pale or inflamed surface, and this chiefly when below the scapula or over the hip. Generally on the legs they are flat, present no inflamed appearance, but show a flat purplish vesication, about the size of a split pea or a sixpence.

Two, three, or even four forms or types of yellow fever have been described by authors. These have been very clearly defined by my friend Dr. Lyons, of Dublin, in the Lisbon epidemic of 1857, which he investigated with so much care and enthusiasm.

The *types, groups, or forms* which he found capable of clinical recognition are,—(1.) The algid form; (2.) The sthenic form; (3.) The hæmorrhagic form; (4.) The purpuric form; (5.) The typhous form.

The first of these, namely, the algid form, is that which presents the most rapid course, and the earliest and greatest amount of prostration of the vital powers. These are the cases which are suddenly killed with the poison. “The patient, while in the enjoyment of his usual health, and in the midst of his usual occupation, feels suddenly the effects, as it were, of a sudden blow from a heavy bar on the back, falls down while walking (or if standing), and dies within a few hours in profound collapse, and after exhibiting more or less of the other symptoms of this fever.” The countenance became sunken, the eye dull and filmy, the surface cold, and the patient felt cold, depressed, and wretched. The face became of a dirty livid hue, and this appearance extended to the trunk and limbs, the surface then presenting innumerable points of minute venous congestion, and sometimes purpuric spots and patches of various sizes. In extreme cases the lips, the breath, and tongue were cold, with a temperature in the axilla not more than 96°, the pulse being small, feeble, and quick; and when the cardiac action became feeble the radial pulse would be obliterated.

The *sthenic form* is a marked contrast to the algid. It is especially well marked in both sexes at the prime of life, and in persons with well-developed muscular frames. Such cases are characterized by well-marked febrile symptoms, severe and persistent headache, much rachalgia at the outset, a high, full, and hard pulse, occasionally thrilling and resisting, with flushed face and throbbing temples. A remarkable elevation of temperature prevailed,—an increase of 3°, 4°, or even 5° Fahr., and in some an increase of 7° Fahr. was observed. Death sometimes took place in a very unexpected manner.

In the *hæmorrhagic type* the cases are the most characteristic and appalling. In them epigastric anxiety, with or without heat, and pain on pressure in the epigastrium, is well marked; but their great characteristic is a tendency to profuse simultaneous effusions of blood from various parts and organs—the *hæmorrhage*

never being single, nor from any one source or organ only. The cases are fatal at an early period ; and all the connective tissue of the body is surcharged with blood. There is less considerable elevation of temperature than in the sthenic form.

In the *purpuric form* the pyrexial state is well marked, with the conjunctivæ and general surface intensely yellow. Purpuric patches commence and spread—sometimes with surrounding œdema. These patches are manifestly caused by subcutaneous effusions of the colouring matter of the blood ; and all varieties and shades of colour and tints are observable.

In the *typhous form* two orders of phenomena may prevail. In one class of cases stupor and nervous depression exist from an early period of the fever, with all the other well-marked and characteristic *typhoid* symptoms ; and to these are superadded the hæmorrhagic phenomena. In another class of these cases the patient, after passing through the sthenic or the algid form, would insensibly glide into the typhoid state, on the cessation of the hæmorrhages (LYONS).

It is of great importance to attend specially to the study of these forms or types ; for, as Dr. Lyons justly observes, much of the discrepancy and apparent conflict of medical testimony on the subject of yellow fever is due to the want of discrimination of those leading characteristics and salient features (LYONS, l. c., p. 375).

When the black vomit is plentiful, or the urine free, the intelligence remains clear and unclouded, but the skin becomes cold and damp, the pulse small, and finally extinct at the wrist, and the patient dies of gradual *exhaustion* and *syncope*.

According to the amount of febrile excitement, the skin is hot and dry, but the experience of Mr. Macdonald leads him to say that in many cases there is a turgid fulness of the vessels, and a tingling heat of the surface, which is imparted in a remarkable manner to the fingers on feeling the pulse. This feverish heat of skin Mr. Macdonald thinks is much more moderate in cases treated in the open air than in those treated in the wards of an hospital, however well ventilated.

There is great irregularity in the temperature of the surface. Sometimes the forehead is the hottest part of the body, occasionally it is the chest. The uncovered parts, in the latter stages of the disease, are easily reduced in temperature, and thus, while

the exposed chest and extremities may feel cool to the touch, the axilla may raise the thermometer to 102° or 103° Fahr. The highest temperature Dr. Blair has observed was 107° in the axilla.

Mr. Macdonald notices that the pulse is at first quick, and of considerable strength, though still compressible, and may vary in these respects within certain limits until it becomes feeble. In those cases which have proved rapidly fatal there has been a marked gradual decrease in its strength; and finally, when the ferrety eye grows clear, and a pallor of countenance shows the mischief of blood exudation going on within, it is scarcely to be felt.

Observations made on the urine, in yellow fever, by Dr. Blair, show that it is always acid in the first stage, and continues so generally till convalescence, when it becomes alkaline, or until it becomes heavily charged with bile. During the early stage the urine is normal in colour, clearness, and quantity. About the third day the colour alters, and becomes of a sulphur, primrose, straw, or light gamboge hue, perhaps slightly turbid, and with a little floating sediment. The colour deepens till it becomes yellow or orange; and if the case ends in convalescence, the urine is copious, and may appear black. Sometimes the urine has a pale, watery, smoky appearance, with a layer of blood-corpuscles in the sediment, and sometimes it is very bloody.

Albumen appears on the second or third day generally; in some cases as early as the first day; and in a few cases not till the day of death, and after black vomit has set in. Albumen appeared in every fatal case of normal duration. It sometimes ceased suddenly in convalescence, and always before the yellow suffusion of skin and eye, or bile in the urine, had disappeared. Between the eleventh and twentieth day of grave cases it generally disappeared. Its colour was never white. When the urine appeared turbid, it was due to the presence of mucous epithelial matter, coagulated albumen, coats of the urinary tubuli, or fine capillaries of the kidney or mucous membrane passed out with the urine. The tube-casts are generally short, thick, club-shaped, and opaque, attended with large organic cells and epithelial scales. Crystalline deposits are rare. In females the catamenia are sure to appear, whether due or not. No sign is so dooming as a suppression of urine, *black vomit* not excepted.

The alvine evacuations may be black towards the close of the disease, or very dark green, and bilious; but after the black stools have ceased, they are succeeded by evacuations which resemble fine, dark, sandy mud, and named the "caddy stool." As the disease still further advanced, and towards its fatal termination, the dejections again changed their character. They became scanty and mucous, of various consistence and colour. These mucous stools almost always appeared *after* black vomit, and were contemporaneous with the scanty urine before described. The alvine evacuations in yellow fever, from the beginning to the end of the attack, are always alkaline, except in one instance, that of the black vomit stool: in that it is always acid. Its chemical quality is evidently due to the admixture of a portion of the black vomit, which has descended (if not found in the intestines) by peristaltic motion into the intestines, and mixed with the scanty mucous stool, and in such quantity as not only to neutralize it, but to be in excess. The scanty thick mucous stool—almost a jelly—has generally a little thin serum around it in the bottom of the pot. The bulk of all these varieties of the scanty mucous stool consists of mucus, broken-up epithelial matter, and myriads of epithelial granules. Sometimes little wavy flakes, like morsels of cuticle, are also to be found. They also frequently contain the crystalline bodies of the caddy stool, particularly when they are rather thin and serous. By appearance, they would be taken for rectal stools, and the results of tenesmus; but such is not the case. A burning sensation is often complained of, but seldom any tenesmus, and no doubt these stools consist of that mucous matter which we find after death lining the intestinal canal generally. In a few cases where there has been total suppression of urine, these stools have become diarrhœal.

The first ejections from the stomach of a yellow fever patient are seldom seen by the physician. Mucus and bile soon appear, occasionally with a streak or speck of blood, and with violent retching. The ejections are alkaline. Generally after the first vomiting the stomach becomes tolerably settled, until the second stage sets in, on the second, third, or fourth, or as late as the fifth day of the disease. Then, without warning or nausea, but on any trifling provocative, the stomach suddenly ejects a quantity of clear, pale, limpid, or slightly opalescent *acid fluid*—the *white*

vomit, which indicates the beginning of the stage of acid elimination, and is generally cotemporaneous with the first shedding of epithelium from the tongue. Sometimes the evacuation of this vomit has a critical effect, equivalent to the perspiration of intermittent fever. True white vomit consists of serum, more or less acid, which remains clear on the application of heat and nitric acid.

The transition of symptoms from *white* to *black vomit* is generally gradual; and is attended with a "suspicious sediment" of "snuff-like specks" before it merges into well-defined *black vomit*. The stage of acid elimination continues to the close of the disease, and is most intensely manifested during the production of the *black vomit*. The presence of ammonia in black vomit is universal, and may be considered as one of its tests; and its specific gravity 1.004 to 1.006, the temperature of the air being 86°. Its sediment consists of coagulated albumen and the debris of blood-cells. Another test is acidity, and a third is to be observed in the phenomenon that the sediment is dissolved by liquor potassæ, which disengages ammonia.

Another feature in the pathological symptoms of yellow fever may be expressed by the fact that the urea of the suppressed urine is eliminated from the system as a volatile salt, by metamorphosis into a carbonate of ammonia, which as such is frequently found in the breath, in the normal black vomit, in combination with an acid, almost always in the stool, and apparently pervading all the tissues of the body. "It would seem," writes Mr. Macdonald, "as though the mucous membrane of the stomach were called upon to compensate for the defective secreting and eliminating power of the kidneys; and in those cases where little or none of the matter of black vomit was found in the stomach after death, the lining membrane presented a thickened appearance, with a muco-sanguineous coating" (Grant's *Annals of Military and Naval Surgery*, p. 135).

Prognosis.—The data to judge from may be arranged into symptoms which are favourable, and those which are not so. (1.) *The favourable symptoms are*—A slow pulse and moderate temperature of the body, and quiet stomach. Streaks of blood during the stage of black vomit, or after acid elimination has set in, are favourable, if the corpuscles are found entire. If the urinary secretion continue, and the black vomit be scanty from

the first, or is afterwards suppressed, the patient may yet survive. Urine simply albuminous is a less serious sign than when it also contains tube-casts. Free, copious urine, no matter how dark or bilious, is the most favourable of any single sign. Prognostics may be derived from the effects of treatment. (2.) *The unfavourable signs* are—The more fiery crimson the tip and edge of the tongue, the more irritable the stomach, the severer the headache, the worse the prognosis of the first stage, and *vice versâ*; but a streak of blood in the early vomit indicates much danger from the attack. In the second stage the earlier or more complete the suppression of urine, and the more copious the ejections of black vomit, the more imminent the danger. If the urine be scanty, and loaded with tube-casts, entangled in epithelial and coagulable matter, the light buff-coloured curdy sediment before mentioned indicates a complex lesion of the secreting structure of the kidney. It is the urine symptom in its maximum of severity, and is as fatal as if the suppression had already occurred. Blood-corpuscles in the urine are not to be looked on with apprehension. A faltering of the articulation is a bad prognostic, and a difficulty of protruding the tongue enhances it. The danger of the case is enhanced by inflammatory complications, and by hypertrophy of the heart. A recent residence in a temperate climate; the *race* or complexion of the individual; the fact of his previously having suffered from an attack, will enter into an estimate of his chances of recovery.

Prognosis is declared by Robert Jackson to be treacherous and difficult in the extreme.

Treatment.—An early attention to first symptoms among the susceptible is of the greatest value in saving human life.

The diagnosis of cases in which the attack has been said to have been “aborted” by remedies may be questioned, and such a belief is opposed to the doctrines of sound pathology. Moreover, the “heroic” doses of calomel which were given in such cases, combined with quinine, cannot be too strongly discountenanced, for “they were first recommended on the strength of a crazy hypothesis” alone. The practice is said to have frequently proved successful in Jamaica; but, according to Dr. Davy, it was not attended with beneficial results at Barbadoes; and the American physicians at New Orleans have not found it to answer their expectations in stopping the fever.

The large and frequently repeated doses of quinine were often also highly injurious (LAWSON). This discrepancy may in some measure be explained by what has been stated at the outset in explaining the pathology of this peculiar fever. It is in cases where the fever is of the periodic or paludal form, and not the continuous or true yellow fever, that quinine may be of use, if the system can be brought under its influence. The ill effects of quinine in specific yellow fever consist in its checking secretion and deranging the circulation within the head; and Mr. Macdonald considers its employment more suited to the convalescent than to the patient immediately under the influence of the disease.

It is an object to keep the bowels freely open, and to get the skin to act freely. The main object of the physician should be to moderate excessive action in any organ, and to endeavour to bring about as complete a crisis as possible about the fifth day, which seems to be the natural period of resolution of the disease. For this purpose nothing is of more importance than to re-establish the secreting function of the colon, and to obtain fœculent evacuations—not mere bilious discharges—but proper dark-brown fœculent stools (LAWSON). Gentle excitement of an extensive portion of the lining membrane of the colon, with frequent copious enemata of a pint and a half of warm water, in which a table-spoonful of common salt has been dissolved, and to which has been added a table-spoonful of olive oil, or more stimulating enemata, such as of turpentine, deserve a full and careful trial. All the depurative functions must be kept in activity. Turpentine is recommended by Dr. Copland, by Dr. Archibald Smith, and Mr. Laird, of H. M. S. “Medea”—one drachm doses by the mouth, or half-ounce doses as a lavement, several times daily, using it also as an epithem on the abdomen.

“When the mucous surfaces,” writes Dr. Blair, “as indicated by the tongue, were denuded of epithelium, the use of *gum water* was decidedly beneficial. It lubricated, defended, and soothed the raw surfaces. The strength was generally three drachms of the purest powdered gum arabic, dissolved in six ounces of cold water, and a table-spoonful of this given every one or two hours. The patient at last gets tired of it; but for thirty-six or forty-eight hours of the most critical period of the disease it is used without dissatisfaction, and then can be substituted by, or alternated with, smoothly and thin-made arrow-root. When the heat

of surface was ardent, a *wet sheet* or *blanket* was used for the reduction of temperature by evaporation, with frequently very good effect; (and if ice could be obtained, its internal use is well worthy of a trial.) But in the later stages of the disease, when the skin was cool or cold, the patient seemed to have an instinctive craving for its re-application, and frequently asked to be put into it. There would appear to be two causes for this feeling. We find it to exist in cases in which black vomit has been copious, and the associating thirst distressing; also in cases where there has been no black vomit of any consequence, and the breath is highly ammoniacal. In the former class of cases the stomach ceases to be an *absorbing* viscus in anything like the proportion of its secretions and transudations. The skin is therefore employed in reducing the crisis of the blood by the absorption of water, as shipwrecked mariners are said to quench their thirst. But not only does the skin afford an inlet for the imbibition of diluting fluids, but the softening of the cuticle would seem to afford an additional outlet for the noxious elements of the circulation; and it is probably in this direction we must in future look for auxiliary means of relieving the blood of its poisonous, metamorphosed, and effete constituents, the onus of which is now thrown on such vital organs as the stomach and lungs. At one time the heat of the surface was so ardent and persistent that the wet sheet failed to reduce it effectually."

The most distressing symptom in yellow fever, both to the patient and the medical attendant, is irritability of the stomach; it is so constantly present, and so often uncontrollable, that the knowledge of every available means of checking it is of the utmost importance.

The *food* during the course of yellow fever should be of the blandest description—chicken tea, arrow-root, sago, and barley-water constituting the chief articles; and these should be taken in minute quantities at a time when the stomach is at all irritable. This rule applies to drinks of all kinds. The patient is greedy for a large draught of fluids; but by sucking them through a glass tube, or a straw, or a hollow reed of small bore, or by the tea or table-spoonful, they are much more likely to be retained. A cold infusion of oatmeal was found an agreeable drink for Scotch seamen, of which they did not seem to tire. A dislike of sweets was observed among the patients; and when lemonade was asked

for, the usual quantity of sugar was objected to, probably from its rendering the liquid too dense for ready absorption by the stomach, and therefore less quenching. *Tea* was found so uniformly to disagree with the patients, and cause vomiting, particularly in the advanced stages, that at length it had to be expunged from the yellow fever dietary. Dilute alcoholic drinks were given freely, and with good effect. Where brandy could be obtained pure (tolerably free from acidity and fusel oil), and was well diluted with water, that spirit answered every indication. Sometimes the effervescing wines were relished and retained, but they are very liable to the objections of containing foreign matters and the products of mismanaged fermentation (BLAIR).

Mr. John Denis Macdonald has several times proved that four or five minims of chloroform prepares the stomach for the reception and retention of food, by lessening its irritability, and the dose should be repeated a short time before food is again taken, as the effect of the chloroform is transitory. On the same principle the administration of *chlorodyne** may be advocated. Lime-water has been also found to have a most beneficial effect in allaying vomiting, and thus enabling the patient to partake of food; and the essence of beef is well adapted for such cases.

* The composition of *chlorodyne* is variously given by chemists, but the following formula may be given as a very useful one:—

R. Chloroform, *f* ℥iv.; Æth. Sulph., *f* ℥ii.; Theraicæ, *f* ℥i.; Mucilag. Acaciæ, *f* ℥i.; Morph. Muriat., gr. viii.; Acid. Hydrocyanic dil. (2 per cent.), *f* ℥ii.; Ol. Menth. pip., ℞iv. ad vi.; *misce bene*.

Syrup or water may be added to the mucilage of gum, and tincture of cannabis Indica (5 to 20 minims), or other anodynes, may be added at the time of prescribing, if deemed desirable. The difficulty in compounding *chlorodyne* is in getting the *chloroform* to mix with the treacle. It will not do so alone, but the use of a little thin gum, or even water, effects their mixture almost at once. The morphia ought first to be dissolved in the chloroform; then mix with the treacle the gum or water, first using about the same bulk of chloroform and treacle, afterwards adding the rest of the chloroform by degrees, constantly shaking briskly the bottle in which it is made up; and then add gradually the other ingredients. The dose is from 5 to 10 minims, repeated as often as its sedative effects subside (Compare Mr. Squire's *Companion to the Pharmacopæia*, p. 58, and Mr. Ashburner, in *Lancet* for 11th June, 1864, p. 688).

During the course of the disease, *auxiliary treatment* may be required to meet contingent symptoms. This is embraced chiefly in the use of sinapisms and stimulating liniments. Tenderness over the liver may be benefited by them.

Dr. Blair writes that when the primary re-action was violent, and the face was turgid, and the head symptoms severe, arteriotomy was performed, and with benefit. In a few such cases, and when the patient was young, strong, and full-blooded, and where the dynamic congestions were so violent that the vessels yielded to the turgescence and impulse, and blood-corpuscles without tube-casts, or even but a haze of albumen, was present in the urine, a vein in the arm was opened, and free bleeding relieved the tension of the vascular system. In such cases convalescence was slow and unsatisfactory, but the immediate results were beneficial. The severe pain in the loins, which is a constant symptom in yellow fever, is generally associated with renal congestion; and sinapisms or stimulating liniments afford great relief to the lumbar pain (MACDONALD).

With regard to the administration of opium in any form, the rule is, *not to give it when there is suppression, or tendency to suppression, of urine*. Restlessness and sleeplessness are best met by chlorodyne, remembering that the effect of yellow fever on the system is to make it sensitive to narcotics.

The congestion of the kidneys, about the fourth or fifth day, requires watching, so as to diminish the chances of suppression, by reducing congestion and preventing the closure of uriniferous tubes by accumulated epithelium. For this purpose Dr. Lawson recommends cupping, either dry or with the abstraction of blood, and the use of frictions, with stimulating liniments over the loins. These, with warm baths or hot-air baths, deserve a full trial; and small doses of *acetate of ammonia*, with *potash* or *soda*, or their salts in common use, with diaphoretics (so as to act gently on both kidneys and skin), may prove beneficial.

SECTION III.—THE LITORAL, MALARIAL, OR PALUDAL FEVERS.

Characterized by one or other of these names, three varieties or forms of fever are understood to exist, having many essential features in common. These are *intermittent fever* or *ague*, *remittent fever*, and that variety of *yellow fever* characterized by

periodicity of febrile recurrence. The specific or contagious form of *yellow fever* is now understood to be a different fever from remittent and intermittent fever, and it has been considered separately in this text-book as a continued fever *sui generis*. The malarious form of *yellow fever* is, on the other hand, the same in kind as the intermittent and remittent fevers, but varies greatly in the extremes of its severity. So great, indeed, are the differences induced by the common malarious poison, that "if any one had seen only the milder forms of *remittent fever*, and had no opportunity of tracing up its several grades, he might well believe, when he saw suddenly the severest variety, that he had before him a distinct affection" (ALISON). Such a belief is entertained by not a few. All of the three fevers, however, which are now about to be considered are similar pathologically; while all seem to take their origin from terrestrial aëriiform emanations, which are sometimes rendered more active or dangerous in connection with human beings congregated together, and in certain relations as to physical climate, and particularly as to temperature.

Pathology.—In these forms of fever a malarial poison of an unknown kind, generated in paludal regions or litoral districts, is absorbed, and affects the blood, as cholera, typhus, and other miasmatic poisons do. The poison, in the absence of any better name, is known as "*malaria*;" and as physicians have merely inferred the existence of such a poison, no exact knowledge has yet been obtained as to its nature and source. Indeed, it still remains to be shown that *malaria* have a substantial existence. No poisonous principle has yet been chemically demonstrated in the air of malarious regions. But many other acknowledged disease poisons are in a similar predicament as to proofs of their substantial existence; and the general impression with regard to *malaria* is, that it is presumed to exist as a gaseous fluid in the atmosphere of certain regions.

After a period of latency, more or less long, functional disorders of the great nervous centres are brought about, terminating in the phenomena either of intermitting, remitting, or yellow fever. These fevers may exist without any alteration of structure being set up, and the patient often dies from the severest forms, with hardly a trace of disease being discoverable. In the milder forms of these fevers, however, a greater number of organs and

tissues are morbidly altered than perhaps in any other disease, as the liver, spleen, lungs, heart, brain, and the serous and mucous membranes of the body generally. The specific action of the malarial poison, within certain limits, may be said to be in the inverse ratio of the intensity of the fever which attends its action. The affections of the liver and spleen also vary greatly according to the country; for in some parts of India the spleen is the organ chiefly affected, while in other districts it is the liver; the nature of the country, perhaps of the soil, impressing evidently some peculiar character on the poison.

The patients labouring under intermittent fever in this country generally recover under medical treatment, without any manifest derangement either of structure or of function of any organ or tissue. When, however, the disease is neglected, the liver may suffer, the disordered function of that organ being generally indicated by jaundice; or inflammation of the liver may ensue, of which jaundice may or may not be a symptom; and this inflammation may be acute or chronic, diffuse or limited to one place. If a liver, previously healthy, becomes the seat of diffuse inflammation, it is of the deepest hepatic tint, and loaded with blood; and we find it often greatly hypertrophied, filling the abdominal and pelvic cavities, and, according as the inflammation is acute or chronic, either greatly indurated or so softened as to be easily broken down. In a few instances this inflammation may terminate in abscess, generally of the usual phlegmonous character. On the contrary, if the liver be previously diseased, its colour, even when the seat of abscess, or otherwise most acutely inflamed, may be of the palest yellow, and its texture sometimes so soft and broken down that the larger blood-vessels may be dissected out with the fingers, or so indurated as to form a shapeless mass of varying magnitude. When abscess forms, it may rupture into the duodenum, or into the cavity of the abdomen, or it may point externally.

The paludal poisoning also often produces structural alteration of the spleen. In these cases that organ has been found sometimes so enlarged as to weigh from ten to thirty pounds, greatly exceeding the liver in size (ague cake), while in other cases it is sometimes even less than natural. In consistency, also, it varies from a state of almost fluidity, a mere bag of blood, to a hardened mass with a distinct indurated edge. It is sometimes the seat of

abscess; or its parenchyma is transformed into a soft mass of pigment of an extremely dark colour.

The functions of the peritoneum may be alone deranged, so as to produce dropsy; but every form of peritoneal inflammation may precede or accompany the *ascites*,—as the serous or the purulent, with diffuse or partial local adhesions.

These are the most usual alterations of function and of structure in the mild paludal fevers of the present day; and in estimating the relative frequency of these secondary affections, *ascites* is the most common, then *jaundice*; while *peritonitis*, *hepatitis*, and *splenitis* are less frequent, and occur, perhaps, in nearly equal proportions.

The pathological phenomena which attend severe intermittent and remittent fever are much more severe, and extend over a greater number of organs. The information afforded us by the dissections of Davis, and the observations of Sir Gilbert Blane, in the cases of the Walcheren remittent; of Jackson in those of the West Indies; of Burnett in the Mediterranean, enable us to understand at least the tendency of the morbid action. Sir Gilbert Blane, in his observations on the Walcheren fever, remarks that the structural derangements were more frequent (especially swelling of the liver and spleen), which then occurred in a very few weeks. Such results seldom occur in England, except under a long continuance of the disease, or after frequent relapses. The morbid changes also extended to the mucous membrane of the stomach, which, in a few instances, was inflamed and ulcerated, and the ulcers had generally a sharp perpendicular edge, as if made with a punch. In cases which died dysenteric, the large intestines, and more particularly the sigmoid flexure and the rectum, were always much contracted, thickened, inflamed, and ulcerated; the ulcers being often so numerous and so confluent that the whole inner surface of the gut appeared in a state of granulation. There is a marked tendency in the phenomena of these paludal fevers to become inflammatory, the congestion of some organ proceeding at once to exudation from the blood-vessels into its parenchyma, which appears to be the cause of prostration and of fatal results. "The significant term *bilious*," writes Sir Ranald Martin, "as applied to these fevers of the East, is not an accidental or a misapplied term, as modern statistics fully show. A severe disturbance of the hepatic

function is almost universal in the progress of the remittent fevers in the East."

There is another remarkable tendency to be noticed in the persistent effects produced by intermittents—namely, that they impress a character of periodicity to subsequent ailments, especially neuralgic affections; and the disposition to the recurrence of these diseases seems to last for life. Susceptibility to the action of the paludal poison does not diminish, but rather increases by continued residence where it prevails. The returns published by the War Office and Army Medical Department show such a result in the West Indies. Thus, while the annual mortality among the troops resident one year in Jamaica was 77 per 1,000 mean strength, in those resident two years it was 87 per 1,000, while of those still longer resident it was no less than 93 per 1,000.

"In making calculations of efficient force," writes Sir James Macgrigor, "this description of men could not be relied on for operations long continued in the field" (speaking of men who had suffered from an attack of paludal fever), for "we found that in those who were convalescent or lately recovered from ague, the causes next prone to reproduce the disease were exposure to a shower of rain, or wetting the feet, full exposure to the direct rays of the sun, or to cold, with intemperance, irregularity, or great fatigue. There are many instances also of the same person being repeatedly attacked with the West Indian malarious fever. Sir Ranald Martin writes, with regard to himself, that "after a residence of ten years in Europe, I happened to pass three nights at the best hotel in Strasburg, at a time when ague prevailed in the garrison amongst the French soldiers who had served in Algeria; and two days after quitting the town I was seized with ague at the hour of eleven A.M. (the hour at which ague used to commence with me in India), and I was the only person of the party who was so affected."

The opinion regarding the pathology of *yellow fever*, which holds that it is "*an intense form of the bilious remittent of the tropics*," has given rise to much discussion.

The investigation of this point, however, is attended with extreme difficulties, and is to be carried out with reference to two questions especially,—namely, (1.) The type or mode of progress of the symptoms in mild and severe cases, compared

with cases of remittent fever in all grades of severity and stages; (2.) The pathological characters of the morbid processes which take place in severe forms of yellow fever, compared with those of remittent.

Three opinions have thus been held regarding the essential nature of yellow fever. These are,—(1.) That there is a *malarious form of yellow fever* which is an intense and virulent form of remittent, and which becomes more or less a continued fever (CLEGHORN, LIND, HUNTER, ALISON, CRAIGIE, MARTIN). (2.) That it is a continued fever of a specific kind, different from all other continued fevers (CULLEN, CHISHOLM, BLANE, WOOD, HIRSCH, ARNOLD, MACLEAN). (3.) That it is a mixed fever, of a type variable between the remittent and continued forms (JACKSON, MOSELEY).

The grounds upon which the first of these opinions is accepted are—that in the symptoms and effects, progress and pathology, of remittent fevers and ordinary cases of yellow fever, we are unable to discover any essential differences, but merely what is due to intensity of morbid action, degree, and rapidity of progress. Comparisons have been drawn in this way between the summer and autumnal *remittents* of the south of Europe, the *remittent* fever of the Mediterranean, the tropical *remittent* of the East and West Indies and Central Africa, the Bulam fever, or the fever of Sierra Leone and Fernando Po, on the one hand, and between some of the cases of the *yellow fever* of Cadiz, Gibraltar, Malaga, Carthage, Leghorn, Vera Cruz, Havanna, Jamaica, St. Domingo, the West Indies generally, and the United States, on the other. There are also cases in which no distinction can be drawn between the symptoms, the effects, or the rapidity of action, if the case (considered to be a remittent) is compared with some cases of so-called yellow fever. In other words, it is not possible to distinguish some cases, and say with certainty that they are cases of remittent rather than of yellow fever, or of yellow fever and not remittent.

Causes and Modes of Propagation.—Facts tending to establish the concurrence of certain terrestrial, gaseous, or meteorological phenomena, as necessary to the generation and development of these fevers, are of a very conflicting nature. The concurrence of some, however, are sufficiently obvious, and are applicable to the litoral and paludal fevers generally.

By numerous observations it has been established that some aëriform material of a poisonous nature is exhaled from marshy or wet grounds in the progress of drying. Agues have always been observed to be the diseases of moist or marshy districts; and to prevail most in low, swampy, and humid countries, where seasons of considerable heat occur. The vicinity of marshes, or of a district that has at some recent time been under water; the banks of great lakes, and the shores of great rivers and seas, where the water flows slowly, and in some places stagnates, in shallow rivers over land alluvial, low, and flat; extensive flat tracts of wood, where much moisture is constantly present, where the process of drying is uninterrupted, and yet the surface constantly exhaling humidity;—these are some of the terrestrial physical conditions in which the paludal and the litoral fevers are found to abound. It must also be admitted, however, that these diseases do not prevail in *all* marshy districts, and they cannot, in some cases, be traced to a residence in the vicinity of marshes. Dr. Wood gives an interesting example of the occurrence of ague from an *irritant cause*, combined with the force of habit. “For seven successive nights M. Brachet bathed, at midnight, in the river Saone, towards the close of October, when the water was cold. Retiring to bed after each bath, and covering himself warmly, considerable re-action took place, which terminated in perspiration. At the end of the seventh day he ceased to bathe, but was, nevertheless, nightly, about the same hour, attacked with a regular intermittent paroxysm, consisting of the cold, hot, and sweating stages, which returned for about a week, when it ceased spontaneously on the occurrence of an event which kept him out of his bed at the hour of paroxysm, and induced him to take a ride on horseback, which excited and warmed him.” Cases having their origin in such causes, however, are of exceedingly rare occurrence, so far as the records of medicine show.

The concurrence of circumstances under which paludal and litoral fevers have been observed to become developed may be shortly stated as follows:—(1.) A certain degree of heat. A high temperature is especially favourable to the production of malaria, and the more so when acting on moist alluvial soil. (2.) A certain relation as to season, variable with the geography of the locality in which such fevers prevail. The season of the year

most marked in tropical climates is that which immediately succeeds the cessation of the rains, or; as it is called, "the drying up of the rains." (3.) Low swampy grounds and extensive rice fields are well-known sources of malaria. In such districts clouds of mist are often seen, wafted along the earth's surface for miles; and it is believed that malaria, whatever be their nature, cling to such mists. But although it has been observed that absolute marshes do not always produce agues, nor that agues are always due to obvious marshes, yet it is generally found that in districts where such paludal fevers abound the surface is porous, penetrable, and retentive of moisture, although it does not appear on the surface of the ground; that the district had been at one time submerged; and that it continued slowly but constantly to undergo the process of desiccation: or while at certain seasons it imbibes moisture from local or meteorological sources, at other seasons it undergoes the drying process under intense solar heat. Such are some of the most sickly and febriferous districts in Europe, India, and America. For example, the Maremma of Italy; the district of the Lakes near Varna, in Bulgaria; many districts in Burmah; many newly cleared tracts in North America; and many parts in the south of Spain. In most of these places the conditions of the surface of the ground are very much alike. While no obvious appearance of a marsh exists, the vigour of vegetation is extreme, amphibious animals abound, of the batrachian kind, plants and cephalapodous mollusca of notoriously marshy regions find a habitat, and the rich alluvial soil is so imperfectly cultivated that the process of vegetation is not adequately exhausted, and a surface of humid ground is exposed to the solar heat, and so exhales a material which exercises a persistent deleterious influence on the human frame. It is believed that the number of insects and some reptiles with which a place abounds are more significant of its insalubrity than almost any other circumstance; and that a mixture of animal and vegetable matters undergoing decay give rise to miasms much more noxious than those resulting from vegetable matter alone. Dr. Fergusson, in *The Edinburgh Philosophical Transactions*, vol. ix., p. 273, was the first author who clearly proved that the drying of all porous soils, from which watery fluid readily evaporated, was the genuine source of exhalations capable of producing the paludal fever; and that the febriferous activity

of these exhalations was influenced by the character of the season, the moisture, the temperature, and the aërial movements of the atmosphere.

The evidence regarding the geological nature of soil as a cause of ague is somewhat conflicting. It is a fact that the usual localities in which paludal fevers abound are those in which the soil consists of mineral, vegetable, and animal matters, mixed together in such proportions and of such constituents chemically as tend to absorb moisture and retain it, and subsequently to decompose. Such soils are known as *alluvial*. Paludal fevers abound, however, where soils of a different nature predominate. Level plains of sand, or dry, loose, open gravel, are soils where malarial fevers have prevailed.

“The first time I saw intermittent and remittent fever become epidemic in an army,” writes Dr. Fergusson, “was in 1794, when, after a very dry and hot summer, our troops in the month of August took up an encampment at Rosendaal, in South Holland. The soil was a level plain of sand, with perfectly dry surface, where no vegetation existed, or *could* exist, but stunted heath plants. On digging, it was universally found percolated with water to within a few inches of the surface, which, so far from being at all putrid, was perfectly potable in all the wells of the camp.”

High grounds near exposed marshes are often more unhealthy than the places immediately adjoining, which are on a level with them. Rocky places, such as Ciudad Rodrigo, Gibraltar, and Malaga, have now and then been ravaged by epidemics of litoral and paludal fevers, and the rocky shores and islands of the Mediterranean—for instance, Minorca, Sardinia, Sicily, Cephalonia, and all the Cyclades—abound as much in these fevers as the most level parts of Holland; and the West India Islands, most of which, although coralline rocks, are the native soil of these diseases. Soil composed of tenacious or stiff clay (argillaceous) is highly retentive of moisture, and is difficult either to dry or to drain. The basin of the Thames, comprehending Middlesex, Essex, Surrey, and Kent, is almost entirely clay land, and is the district of England where agues most of all prevail, especially along the banks of the Medway and the Thames. In the days of Sir Gilbert Blane agues had almost entirely ceased to occur in London, and the cases which he treated he believed to have been imported from malarious districts around; and the same may be

said of those of the present day. A hundred years before the time of Sir Gilbert Blane, however, we find that agues prevailed in situations in the town of London where they are now wholly unknown, such as Russell Street, Covent Garden, Fleet Street, Fetter Lane, Newgate Street, Paternoster Row, Cheapside, Smithfield, and Fenchurch Street. At the present time ague rarely occurs in London, except on the south side of the river, especially in Bermondsey and Rotherhithe, and chiefly in persons who have recently been exposed to malaria in Kent or Essex, and who have come from marshy districts, either quite recently or within a few months. The malarious influence, still in the metropolis, seems, however, sufficiently powerful to imprint a periodic character upon various local affections, and occasionally to give rise to fevers of a remittent type. Recently (in 1856) such affections have been unusually prevalent; but the forms of ague now met with in London are more tractable and milder than those which formerly prevailed (DR. PEACOCK).

It is observed that the surface of the earth may be dried either by the direct rays of the sun, or by currents of hot dry air wafted over it, or by both combined; but it is principally by the direct rays of the sun that the deleterious material of the soil is liberated; and it seems to be at a certain period of this "drying up" process that the exhalations are more potent than at another time in developing paludal fevers. The exposed grounds, after clearing off the copious vegetation from dense jungles, so as to admit the influence of the sun's rays in "drying up," is known to be a fertile source of malaria.

There appears also to be a certain state of the human frame which renders it more than usually susceptible to this disease. The natives of warm and tropical climates are much less frequently and less violently attacked with paludal or litoral fevers than settlers or visitors from other lands, such as the natives of Europe or the northern parts of America. In the Mediterranean, along the coast of Africa, in the East Indies, in West India Islands, in the Southern States of the Union, new-comers from the northern latitudes are almost invariably attacked, and suffer much more severely from the fever than those who have been long in the country. It has been also noticed that those who, after residing in a territory where paludal fevers abound, have been out of it for some time, an augmented susceptibility to

renewed attacks of the fever becomes manifest on their return (CRAIGIE).

Other causes predispose to those fevers, and none more than laborious or fatiguing duty in military or naval operations, labouring in the sun, excess in eating or drinking, intellectual exertion combined with bodily fatigue, and a crowded state of the population. Indeed, *sun-stroke*, or heat apoplexy, is regarded by many as a form of remittent fever (JOHNSTON, MARTIN, HILL).

When a remittent fever, or other paludal or litoral fever, has, under certain concurrent circumstances of weather, season, and physical peculiarities, made its appearance in any locality, it necessarily attacks all those who are by constitution, habit, and age, susceptible and predisposed; and the majority of these, especially if enfeebled by previous dynamic or organic disease, it destroys. The population, therefore, which outlives such an epidemic visitation are no longer equally susceptible, and are greatly less likely to be attacked the ensuing season, unless it is more febriferous than the past, which, though sometimes, is not generally the case. The effect of this, therefore, is, that while the endemial disease continues for a season to attack and destroy its *ordinary* annual proportion of the population, it does not for several years attack the *extraordinary* proportion, because that proportion is not yet ready for, or susceptible of, its attacks. In the course of a few seasons, however, during which the young have grown up and become adult, the adult have become careless, and perhaps irregular and incautious by long immunity, and their constitutions have become less able to resist deleterious or morbid impressions, and the whole population of the place has become generally augmented by the arrival of persons from various other countries; a considerable number of susceptible persons is gradually accumulated; and at the end of five or six years a place of 25,000 or 30,000 inhabitants becomes augmented perhaps by an additional fifth, or even by a third. The majority, or the totality of these persons, are all more or less predisposed and susceptible; a season of excessive drought ensues, in which solar desiccation and little wind form conspicuous characters; fever appears, and spreads at first slowly and gradually, but afterwards, springing up in many points, rapidly coalesces; and in a short time is so general and fatal that it assumes an epidemic character. The usual mortality in the meantime takes

place; all the susceptible and predisposed subjects pass through the disease or are cut off; and the population of the place is once more reduced to its state of epidemic insusceptibility and endemic or ordinary liability. This is the usual course of epidemics of paludal or litoral fevers in all countries within the tropics, and, indeed, within the 45th degree of north and south latitude (CRAIGIE).

A question of much interest in connection with *malaria* has given rise to considerable speculation. It relates to the *varieties* of the malarious poison. Is the poison which gives rise to a *quotidian ague* different from that which produces a *tertian*; and is this, in its turn, different from that which produces a *remittent fever*; and this, again, different from that which gives rise to the malarious form of *yellow fever*? Our knowledge is not yet sufficiently precise and extensive to settle these points. But when we see a large body of men placed under the same circumstances as to food, drink, and clothing, and labour, and exposed to the same causes of disease, in the same way and in the same place, some of whom are seized with *quotidian ague*, others with *tertian*, and others with *remittent fever*, the presumption is that the same cause has produced different diseases, according to its intensity, the constitution of the individual, and the predisposing causes to which he may be subjected. Other facts also favour this presumption—namely, *remittent fevers* are known to pass into *quotidian agues*; and these again into *tertian agues*.

AGUE—SYN., INTERMITTENT FEVER.

LATIN, *Febris intermittens*; FRENCH, *Fièvre intermittente*; GERMAN, *Kaltes Fieber*—SYN., *Intermittens*; ITALIAN, *Febbre intermittente*.

Definition.—*Febrile phenomena occurring in paroxysms, and observing a certain regular succession, characterized by unnatural coolness, unnatural heat, and unnatural cutaneous discharge, which prove a temporary crisis, and usher in a remission. These phenomena are developed in an uninterrupted series or succession, more or less regular, which pass into each other by insensible steps.*

Symptoms.—The disease may be sudden in its attack, and without previous illness; but more commonly it is preceded by general indisposition, headache, weariness, pain in the limbs,

thirst, loss of appetite, white tongue and frequent pulse, high-coloured urine and dark-coloured discharge from the bowels. These *prodromes* are accompanied with well-marked exacerbations and remissions of fever, displaying a periodic tendency. After this feverish state has lasted from four days to a fortnight, the patient is seized with severe rigor, and the ague is manifested. The phenomena of an attack or "fit of the ague" are the following:—

The paroxysm, like the disease, may be of sudden invasion, and the patient may be in good health up to the time of attack; or it may be preceded by languor, debility, frequent yawnings, and great unwillingness to make the least exertion. In whichever way the cold stage begins, the patient experiences first a sensation of coldness of the extremities, then of the back, and lastly of the whole body; at the same time the nails turn blue and the features shrink, becoming pale and sharp. If the case be severe, the whole body shrivels up, turns purple, and the surface of the skin assumes that rough condition popularly named "goose-skinned." The coldness increasing, the motor nerves of the fifth pair are affected, and the teeth begin to chatter; and this tremor extends to every muscle, till the whole body shakes with rigor. Cough, dyspnœa, and oppression of the præcordia now occur, with a painful sensation round the temples and down the back. The patient often suffers from nausea and vomiting, and the latter symptom is speedily followed by the hot stage. When the cold stage has lasted a period varying perhaps from half an hour to two hours and a half, a re-action takes place, accompanied by partial warmth, or flushings. These extend, and at length the whole body acquires a heat greater than natural, or from 105° to 107° . As the heat returns, so also does the colour, and the body, especially the face, becomes preternaturally swollen and red. The hot stage being formed, the heart and arteries beat with unusual violence, and headache, with a frequent full pulse, and all the distressing symptoms of continued fever, are present. "The mean duration of this stage is from three to eight hours. At its close a gentle moisture breaks out, first on the forehead, and thence extends till the patient lies in a general sweat, sometimes so profuse as to soak the bed and linen as completely as if they had been dipped in water. After the sweat has continued to flow for some time the fever gradually abates, a state of apyrexia ensues,

Cold stage - 10 min - 8 hours
Hot stage $\frac{1}{2}$ hr - 24 hrs
Sweating shorter than either

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the paroxysm is terminated, and (a sense of exhaustion excepted) the patient feels restored to health. Sometimes, however, he continues pale, debilitated, and incapable of all exertion, till, on the recurrence of the paroxysm, the symptoms just described are repeated.

Pulse Upon the approach of the attack the pulse is slow and feeble, but as the sense of coldness increases it becomes small, rapid, and irregular. When the hot stage forms, the pulse becomes full and strong, and on the sweat breaking out it again becomes soft, less rapid, and at length natural.

The tongue, in mild forms of the disease, is clean in the cold stage, white in the hot stage, and again cleans after the sweat has flowed. In severe cases the tongue is white during all the stages, and also during the apyrexia, while in the worst cases the tongue is brown in all the stages. Excepting some unusual instances, attended throughout with diarrhœa, the patient seldom passes a stool till towards the close of the paroxysm, when it is generally a loose one. It frequently also happens during the cold stage that tumors subside, or ulcers dry up, but the tumor generally re-appears, and the ulcers discharge as soon as the sweating stage is formed.

The paroxysm of intermittent fever, of whatever description, is conventionally considered to terminate in twenty-four hours. The duration, however, varies in different types. These types or varieties have been named—*quotidian*, *tertian*, and *quartan*; and of these there are sub-varieties—namely, *double tertian* and *double quartan*. The mean length of a *quotidian* paroxysm is about sixteen hours, that of a *tertian* ten hours, and that of a *quartan* six hours.

X The febrile paroxysm, or fit of intermittent fever, has three stages—a cold stage, a hot stage, and a sweating stage. These three stages are not necessarily of an equal duration, but vary greatly in different cases. The duration of the cold stage is from a few minutes to five or six hours, and in general, if the case be severe, the shorter the cold stage the longer the hot stage. The hot stage may last from half an hour to any period less than twenty-four hours. The sweating stage is generally shorter than either of the former, and sometimes does not exist at all. The rule, however, is, that the *quotidian* has the shortest cold stage and the longest hot stage; the *tertian* a longer cold stage and a

shorter hot stage than the *quotidian*; while the *quartan* has the longest cold stage and the shortest hot stage of all the varieties. An "*irregular*" variety is also recognized, which takes the form of "*Brow ague*."

The varieties of intermittent fever are distinguished from each other by the interval of time which elapses between each paroxysm. For instance, when the paroxysm returns every twenty-four hours it is termed a *quotidian* when every forty-eight hours a *tertian*, and when every seventy-two hours a *quartan*; and these primary types have been extended by early writers to every period comprised within a mensual or bimensual period.

Of these primary types it is believed that in Europe the *tertian* is by far the most common type, then the *quartan*, and lastly the *quotidian* (WATSON, COPLAND, CHRISTISON). But this law is by no means general; for M. Maillot treated 2,354 cases of intermittent fever occurring in the French army in occupation of a portion of the northern shores of Africa, and he found of that number 1,582 were *quotidian*, 730 *tertian*, and 26 *quartan*. In the Peninsular war the *quotidian* was likewise the prevailing type, and at one time they were in the proportion of 16 to 1 of any other type. In the West Indies the *tertian* and the *quartan* are only about one-twelfth of the whole number of intermittents treated, the rest being *quotidians*. At Prome, in Burmah, 298 cases from the 2d Bengal European Regiment were admitted into hospital for intermittent fever in 1853, of which 249, or 83·5 per cent., were *quotidian*, and 49, or 1·6 per cent., were *tertian* (MURCHISON). The results given concerning the Madras Medical Service are very similar (WARING).

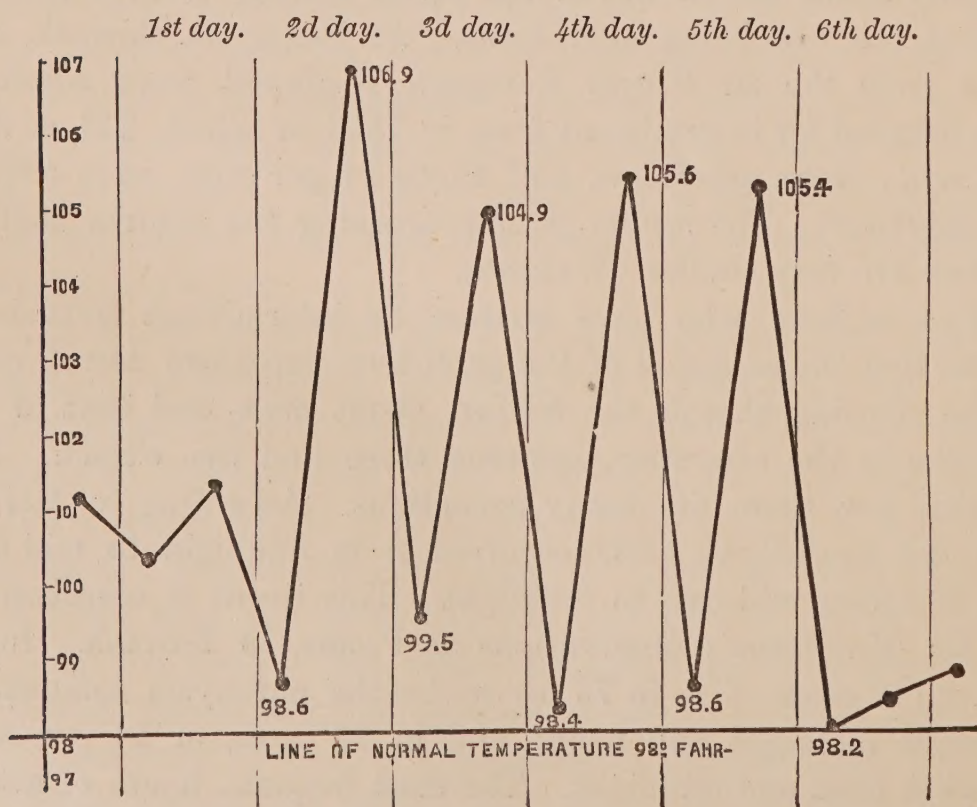
Most authors who have written on intermittent fever have stated that the accession of the *quotidian* paroxysm occurs early in the morning, that of the *tertian* about noon, and that of the *quartan* in the afternoon, between three and five o'clock. But to this law there are many exceptions. According to Maillot, of 1,582 *quotidians*, 1,089 occurred from midnight to mid-day, and 493 from mid-day to midnight. This result is corroborated by Dr. Murchison's observations at Prome, in Burmah. In 86 out of 113 cases—*i. e.*, in 76 per cent.—the paroxysm commenced between midnight and noon; and in 27 cases, or 24 per cent., between noon and midnight. The most frequent hours of attack

were 9, 10, and 11 A.M.; and in 65 *per cent.* of the cases the paroxysms commenced between 8 A.M. and noon.

Of 730 *tertians*, 550 occurred from midnight to mid-day, and 180 from mid-day to midnight; out of 26 *quartans*, 13 were seized from mid-day to midnight, and 13 from midnight to mid-day. As the most general conclusion, the paroxysm returned in a great majority of the *quotidian* cases from ten to twelve o'clock, and in the *tertian* from nine to twelve o'clock.

The Temperature in Cases of Intermittent Fever.—The paroxysm of fever, notwithstanding the subjective sensation of chilliness, is invariably indicated by a decided, sudden, and rapid rise of temperature. In this respect it resembles the accession of *febricula*; but while the latter requires only from eighteen to twenty-four hours from the commencement of the rise of temperature to the end of the defervescence, in perfectly normal cases of *intermittent fever* there is a whole day free of fever between every two days of the paroxysm. All the types of the fever present this characteristic peculiarity of a sudden and speedy rise of

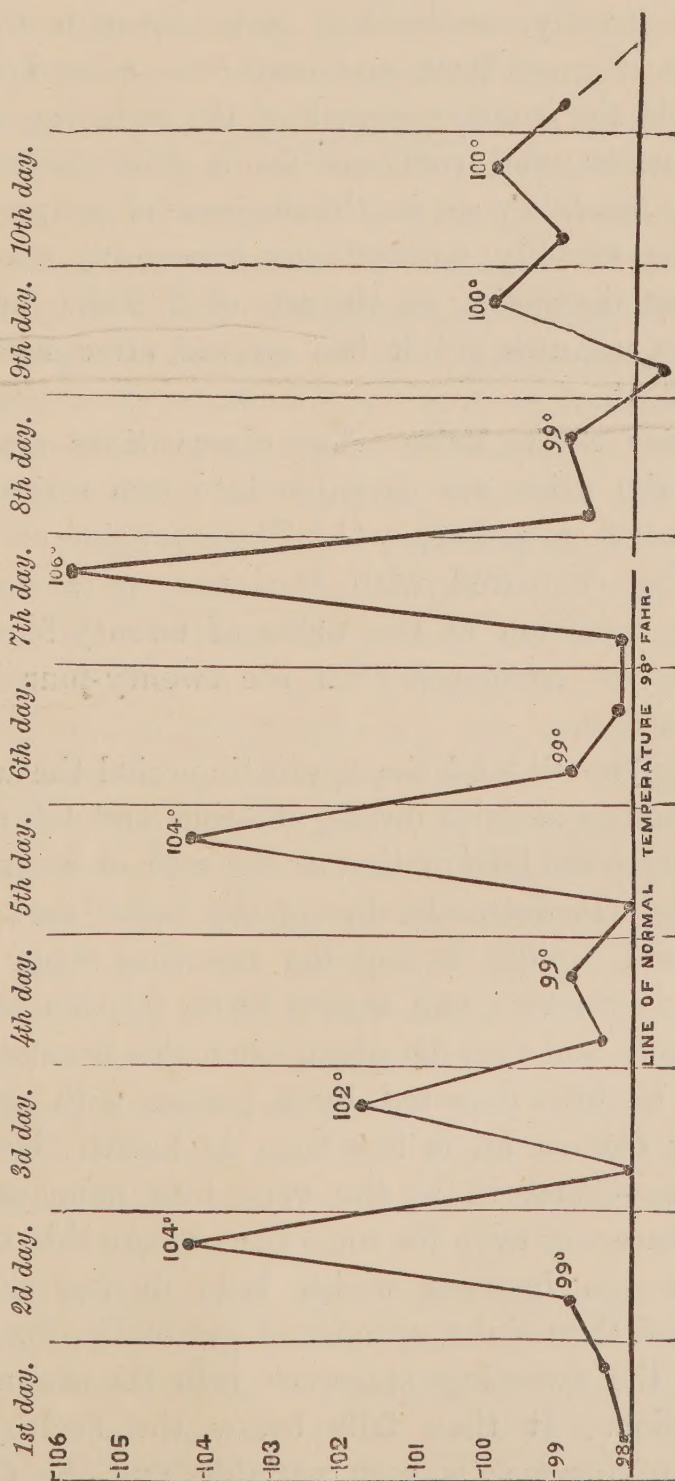
- (1.) TYPICAL RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF *QUOTIDIAN TYPE*. THE RECORDS INDICATE THE HIGHEST AND LOWEST TEMPERATURES DAILY (Wunderlich).



temperature to a high degree (mostly up to 105° or 106.3° Fahr.); and of an equally rapid and complete defervescence, till the period of another fever paroxysm comes about. This comportment as to temperature secures correctness of diagnosis in cases which may be obscure or ambiguous.

The annexed diagrams represent variations of temperature in cases of malarious fever.

(2.) RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF *TERTIAN TYPE*.
THE RECORDS INDICATE THE HIGHEST AND LOWEST TEMPERATURES EACH DAY
(Wunderlich).



223 In a paroxysm of intermittent fever much may be learned as to the relations of the excretions to temperature, and especially those of the urine, by observing the changes of temperature in very short spaces of time: for example, every fifteen, or even every five minutes (MICHAEL, JONES, RINGER). The rise of temperature is found to begin with, or even to precede, the sensation of chilliness. It takes place at first slowly; and gradually, by about the middle of the period of chilliness, the rise becomes greatly accelerated, lasts through the period of the sensation of great heat, and may even extend into the sweating stage. At the commencement of the sweating stage, small vacillations occur, and continue for a short time; and when the sweating has fairly set in, the decrease of temperature begins, and progresses steadily, without any temporary rise, and with great regularity, decreasing at the rate of 2° Fahr. (or more) every five to fifteen minutes, till it has arrived, after several hours, at the normal heat.

Condition of the Urine.—The observations made on the condition of the urine are divisible into two series, as arranged by Dr. Parkes (l. c., p. 235). (1.) The condition of the urine during the fit, as compared with the urine of a non-febrile period; (2.) The condition of the urine of twenty-four hours during a fever day, as compared with the twenty-four hours' urine of a non-fever day.

223 During the fit and the apyretic period the water of the urine is increased in amount during the cold and hot stages: it is most abundant at the termination of the cold or commencement of the hot stage. It decreases during the latter part of the hot stage slowly, and rapidly during the sweating stage. The amount of increase is variable, and stands in no relation to the quantity of fluid drunk, and may be great when this is small (RINGER). The amount of urea excreted by a person with ague, not actually suffering from a fit, is less than in health; but directly the fit commences—that is, at the very first moment of elevation of temperature—or even for some time before this, the urea suddenly increases—an increase which lasts during the cold and hot stages, and then sinks, sometimes gradually, sometimes suddenly, through the sweating stage, or into the commencement of the intermission. It then falls below the healthy average. The amount of increase is very variable, and the type of the fever

has no influence upon it; but there seems to be a very close connection between the temperature and the amount of urea. The amount corresponding to a degree of Fahrenheit was greater at a high than a low temperature; and in the fit of each day the same amount was excreted for each degree of temperature (*Med.-Chir. Trans.*, 1859). This increase in the urea must be regarded, in some measure at least, as an indication of increased metamorphosis; and the close relation to the febrile heat certainly implies that it owns only this source, and is not caused by elimination following previous retention (PARKES).

The uric acid is greatly increased during the fit; and after the fit there are often deposits of urates, either spontaneously or on the addition of a drop of acid, and it seems probable that the increase in the excretion of uric acid continues for some time after the paroxysm; and the enlargement of the spleen in connection with this great increase of uric acid is probably not fortuitous (RANKE). The influence of quinine in diminishing the amount of uric acid in health is of interest in connection with its effect upon malaria, and with the condition of the spleen in malarious fever (RANKE, BOSSE). The chloride of sodium is increased during the cold and hot stages to a great degree (TRAUBE, RINGER),—to five times the normal amount; and phosphoric acid is diminished to *one-eighth* (NICHOLSON).

The results are contradictory regarding the urine of a fever day, compared with the urine of a fever-free day. This may be explained to some extent by the relative duration of the fit, compared with the fever-free period; and great differences may arise from the comparative length of the apyretic period on the fever days; also from the severity or the reverse of the fit, and from the amount of food and drink able to be taken. With respect to abnormal constituents, albumen is found during the fit in a considerable number of cases. Blood in some quantity, and renal cylinders, are seen about as frequently as albumen; and occasionally chronic Bright's disease is a consequence of ague, (PARKES, l. c.)

Treatment.—The treatment of agues varies in a great degree with the complications of the disease; such as with the splenic and hepatic congestions, and the inflammatory affections of these and other organs, which are apt to be established during the existence of an intermittent fever. During each paroxysm, and

987 11 subsequent to it, the condition of the two important organs referred to ought to be carefully observed; and it ought to be observed, also, whether any symptoms exist of congestion or actual exudation into the cranial or abdominal organs. When the type of the fever is malignant, or of a severe and complex kind, or when the complications are locally severe, it is difficult to cure an ague, which otherwise is a very manageable disease. During the cold stage, especially if it is of long duration, the liver, and especially the spleen, become turgid, the symptoms of which generally disappear with the sweating stage of the fever. It is when the endemic influences are severe, or when the attacks are prolonged over months and years, that these organs begin to suffer permanently from organic disease.

X It is useless to attempt the cure of intermittent fever if the sufferer is permitted to remain within the sphere of malarial influences, or even in those geographical latitudes which are said to be peculiarly malarial. It is now an established fact that none can become acclimated so as to withstand the influence of malaria. When organic complications exist, they must, if possible, be remedied, because they maintain the morbid sensibility during the intermission, and prevent the cure of the ague.

In the warmer latitudes the following account of the treatment of intermittent fever is that laid down by Sir Ranald Martin. During the cold stage of the fever, while emetics seem to be indicated, they are not in repute. Warm drinks, ammonia, ether, camphor, and other diffusible stimuli, with the application of external warmth, seem to be preferred by most practitioners.

(a) During the hot stage a full dose of calomel, with James's powder, should be given at once, and in three hours this should be followed by a brisk cathartic, diluent drinks being freely used meanwhile, along with some cooling diuretic. The tartarized antimony, with nitrate of potash, is recommended, as it answers the double purpose of exciting to action the functions of the skin and the kidneys. On the following morning, the intermission being completely established, the sulphate of quinine is to be administered. The influence of this medicine on a person in health, as observed by Dr. Ranke, is to diminish the quantity of uric acid in the urine (*Med. Times*, May 30, 1857)—an observation which has been recently confirmed by Bosse, of Dorpat, and is of interest in relation to its influence upon the spleen. It

is to be given at intervals of three hours during the day, the patient being kept in bed, and supplied with farinaceous food only. In the simple cases, when removed from the sphere of malarial influence, it may not be necessary to give mercurials more than once or twice, but active purgatives are always beneficial in relieving the full and congested state of the abdomen generally, during the continuance of intermittent fever. There are cases of intermittent fever, however, complicated with hepatic and other engorgements, and which continue to recur despite of all means, until a few doses of calomel, followed by purgatives, are administered; then the quinine, which before failed, will speedily cure the disease. The compound jalap powder, combined with calomel, is found very beneficial for this purpose. It appears that certain morbid conditions both of liver and spleen may produce and maintain the tendency to recurrences of ague. Ramazini relates the case of a patient harassed by an obstinate ague, and who was cured by mercurial frictions administered for syphilis. The influence of splenic disease in keeping up the morbid train of actions of the original fever, and in producing relapses, has been recorded by M. Piorry. In more than 500 cases of ague in which he observed the state of the spleen, he comes to the following conclusions:—namely, that the organ is invariably enlarged during the progress of the fever, and that by the use of quinine the spleen diminishes in size; that its reduction in size bears some relation to the quantity of quinine taken; that the effect it produces upon the fever is in proportion to the reduction of the spleen; that the disease is cured simultaneously with the subsidence of the splenic enlargement; and that the fever is apt to recur so long as the spleen exceeds its normal size.

When the fever is severe, accompanied with præcordial oppression, pain, fulness of the spleen or liver, or both, or where there is severe headache, or headache with giddiness, or an oppressive fulness of the chest, a general or a local blood-letting, or both combined, is imperatively demanded, as a means of promoting cure and preventing future evils. The antiperiodic power of bark, quinine, or arsenic, then becomes more easily developed. According to Dr. Copland, such depletion is almost an indispensable preliminary to the administration of quinine or bark, especially in the complicated and congestive forms of the disease. Without such depletion the medicine will either not be retained, or, if

retained, it will convert congestions or slight forms of inflammatory irritation into active inflammation or serious structural changes. It is chiefly to a neglect of such a mode of practice that unfavourable consequences have so often followed the use of bark, quinine, or arsenic; for their influence is at first to interrupt secretion, or to over-excite, and subsequently to inflame organs already loaded, obstructed, and congested. But if blood is to be drawn at all, it should be drawn at the very onset of the hot stage, or that of re-action; and it should be regulated by the constitution, the age, and the habit of the patient, as already explained.

When, on the contrary, the fever assumes a low adynamic form, or when the patient is anæmic, mercurials must be carefully avoided in the treatment under all circumstances, and reliance placed on change of air, quinine, and chalybeates, and improved diet. With regard to liver complication in such cases, the nitro-muriatic acid is to be used internally, in doses of ten drops, three, four, or five times a day, and externally in the form of baths.

With regard to the doses of quinine, some give very large quantities, such as twenty or thirty grains before the expected paroxysm (MAILLOT); others begin to administer the quinine on the subsiding of the paroxysm, and during the sweating stage. According to the experience of Sir Ranald Martin (which has been great in tropical climates), the most rational plan is to give the quinine every three or four hours during the interval of freedom from fever, and in such doses as the urgency of the symptoms may demand. It is to be administered in solution, dissolved by a small quantity of *dilute* sulphuric acid. He also recommends antimony to be conjoined with the quinine in plethoric subjects; and, on the contrary, if the patient is feeble, irritable, or exhausted, he adds a few drops of tincture of opium to the antiperiodic. When arsenic is given in large doses, and its use prolonged, it permanently injures the circulating system and the mucous membranes of the stomach and bowels. It should be given in small doses, and not persevered in for more than eight or ten days. From six to eight drops of the solution of the College formula may be given every three hours during the interval of freedom from fever. Dr. Murchison's experience in Burmah goes to prove that the practice most effectual in at once checking the paroxysms of intermittent fever is that of

Dr Reynolds gives 3 doses of gr $\frac{x}{2}$
within the interval.
Case Oct 12th to cured thus after quotidian ague for
3 wks.

TREATMENT OF AGUE.

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administering one large dose of quinine during the third or sweating stage. The usual dose given was twenty grains in a draught, with a few drops of sulphuric acid to dissolve the quinine. In no case did he observe any unpleasant symptoms from the physiological action of the drug, although many of the patients complained of slight buzzing in the ears—some amount of which is deemed necessary by Dr. Murchison for the success of the remedy; and when it occurs, "it is a sign that there is no use of pushing the medicine farther." Christison also recommends the administration of large doses of quinine for the cure of tropical intermittent fevers, as deduced from the experience of the medical officers of the Madras army (*Madras Med. Reports*, 1831; *Edin. Med. and Surg. Journal*, Jan. and April, 1855); and Superintending Surgeon Corbyn has long been convinced of the efficacy of this mode of giving quinine (*Indian Annals of Medical Science*, Oct., 1853). Repeated small doses, on the contrary, have been recommended in this country, by many eminent physicians, to be given during the intermissions (HOME, BROWN, BARKER, WATSON); but the evidence of Dr. Murchison is so clear and decided, that one large dose, given as he recommends, seems more efficacious and more economical than repeated small doses.

When the intermittent fever has become chronic, or when there is organic disease of the liver or spleen as a secondary affection, change of climate becomes a measure of necessity, and should never be neglected. Sir Ranald Martin's personal experience does not allow of his writing in favourable terms of the practice of bleeding in the cold stage of ague. After quoting many eminent authorities both for and against the practice, he remarks, "that in Europe, at least, the treatment of intermittent fevers by blood-letting in the cold stage, whilst it has the show of being prompt and energetic, proves, in effect, hap-hazard, systemless, operose, and tedious; and from all that I have seen and heard in the East, the result there has not been more favourable." The rule of practice laid down by Pringle and Cleghorn has received little or no addition in more recent times. Where general blood-letting is had recourse to in the treatment of intermittent fevers, whether simple or complicated, it should, as in the case of all other fevers, be performed at the very outset of the stage of re-action.

REMITTENT FEVER.

LATIN, *Febris remittens*—Idem valet, *Febres pestiferæ singularum regionum*; FRENCH, *Fièvre rémittente*; GERMAN, *Bösartiges endmischtes Fieber*; ITALIAN, *Febbre remittente*.

Definition.—*Febrile phenomena with exacerbations and remissions, the remissions being less distinct in proportion to the intensity of the fever. The fever is malarious, and is characterized by great intensity of headache, the pain darting with a sense of tension across the forehead. It is accompanied by functional disturbance of the liver, and frequently by yellowness of the skin. The malignant local fevers of warm climates are usually of this class.*

Symptoms.—There are so many grades of intensity in remittent fever (varying as it does from a severe intermittent to malarious yellow fever), and so many different modifications are impressed on it, from the great variety of country by which the poison is generated, that it is extremely difficult to generalize the phenomena.

The severer forms of remittent fever may be preceded by certain premonitory symptoms, such as languor, listlessness, restlessness, or chilliness; or there may be a want of appetite, anxiety, lassitude, pain at the epigastrium, pains in the loins and limbs, headache; slow, small, and irregular pulse; coldness of the skin, and chilliness for one or several days before the commencement of the attack: these are symptoms which usher in a short cold stage. But in other cases the attack is sudden, and the patient, for instance, immediately after a hearty dinner, may be seized most unexpectedly with faintness, vertigo, nausea, confusion of thought; and these almost without a rigor, or a very short one, not exceeding half an hour: a hot stage follows, usually of much greater intensity than that which accompanies the worst forms of intermittent fever.

This hot stage, or period of exacerbation, generally commences in the forenoon of the day, or early in the afternoon, subsiding towards evening, or in the early part of the night, the remissions being generally most complete early in the morning. Sometimes, however, the exacerbations come on towards evening, and last all night, the remissions being then most complete in the forenoon; while, in a few cases, there may be two exacerbations in the

twenty-four hours ; and these cases are generally the most severe. The exacerbation is usually marked by much cerebral affection, as severe headache, a painfully acute state of every sense, an injected state of the conjunctiva, and great action of the carotid arteries. The pulse, varying from 90 to 120, is generally at first full, but is sometimes from the first small, and generally soft and easily compressible. The tongue is dry, with a white and sometimes yellowish fur, and a bad taste in the mouth. There is generally unquenchable thirst, parched lips, tenderness at the epigastrium, and sometimes pain, with increased dulness on percussion, in the region of the liver. These symptoms are frequently accompanied by delirium, sometimes of a violent character. When giddiness is distressing, and proceeds to delirium at an early period, and runs high, a severe form of fever may be expected. In other cases the patient is oppressed with great drowsiness, lethargy, or coma. The stomach also is often the seat of great pain and uneasiness, followed by vomiting, and the matters vomited are either colourless, bilious, or bloody. The duration of this paroxysm varies considerably, and when the disease is mild it may terminate in six or seven hours ; but if severe it may last fifteen, twenty-four, thirty-six, or even forty-eight hours ; and Dr. John Hunter once saw a case in which there was no remission for seventy-two hours. Inability to sleep is almost constant. The urine is scanty, high-coloured, and of high specific gravity (1024 to 1030), acid, not coagulable by heat (MURCHISON). Albumen was tested for by Dr. Murchison in numerous instances, but never detected ; and according to Jones's experience in America, it is very rarely present,—a point of difference, if verified, of great importance as a distinction between severe remittents and specific yellow fever (PARKES). In severe remittent fever Jones found the urea increased, and the uric acid lessened till convalescence, when it again increased, and the pigment was also lessened (PARKES, l. c., p. 242). The fever, however, at length remits, sometimes with sweating, but at other times without any sensible increase of perspiration. The first exacerbation is generally the longest, lasting, in some cases, for twenty or twenty-eight hours ; but generally after twelve or sixteen hours the symptoms remit.

The duration of the remission which follows is as various as that of the hot stage. Sometimes it does not last longer

than two or three hours; more commonly it extends to six, eight, ten, fifteen, thirty, or even thirty-six hours. The fever then returns, and in some cases assumes a quotidian type, and has an exacerbation every day, and perhaps nearly at the same time, yet more frequently there is no regularity in the times either of its accession or remission.

X The second paroxysm is always more severe than the first, if the progress of the fever has not been checked during the remission, and usually neither any cold stage, rigor, nor even chilliness precedes it. On the other hand, all the febrile symptoms run much higher, the skin is hotter, the pulse more frequent, the headache greater, the senses more confused, and the delirium or coma, when that exists, more violent in degree and more sudden in its accession. Delirium, with more or less loss of consciousness, may not supervene till the third or fourth paroxysm; and is of a low wandering character in the asthenic form of the fever. The tongue becomes dry, hard, and brown, or almost black; the teeth covered with brownish scales; and the pulse becomes small and weak. These symptoms sometimes persevere with or without the black vomit, till they terminate perhaps in coma more or less profound, great prostration, *subsultus tendinum*, fetid breath, resembling the odour of a dead body, convulsions, and at length in death. The severe forms of the fever are sometimes accompanied with a yellowish hue of the skin and white of the eyes. The yellowness is said to be less where there is a copious bilious diarrhoea, and where the urine is of a dark yellow-brown colour. When the disease does not terminate fatally, amendment is generally observed after the fifth exacerbation, which may subside in very copious perspiration, with the following symptoms of progressive amendment:—the tongue begins to clean and grow moist at the edges, the sordes disappear from the teeth, the thirst diminishes, and the appetite gradually returns. The pulse remains slow and soft, but begins to acquire strength; and the skin continues cool and moist, sleep returns, and the strength is gradually but very slowly recovered. Headache may continue for some days, till relieved by epistaxis. Young and robust men, particularly recruits, who recently arrive in India from Europe, suffer a considerable amount of vascular excitement, with marked symptoms of determination of blood, either to the head or to the

abdominal viscera, at a very early stage of the fever. In such cases the pulse is at first full, and of tolerable strength, the skin burning, and the delirium raging and acute. Again, in other men more advanced in years, or those debilitated by long service in India, by previous disease, or by habits of intemperance, there is very little vascular excitement, even during the exacerbations; and the pulse, though quick, is small and weak. There is no great heat in such cases—there is even coldness of the skin, with a yellow tinge, often severe hiccough and vomiting, with great prostration of all the vital powers. In such cases the chief indications of the exacerbations are increased restlessness, vomiting, headache, or wandering delirium. In these cases the remissions are not well marked, even from the commencement of the attack, (MURCHISON, l. c.)

There are great varieties in the degree of severity and type of this fever, more especially as they occur in England, France, Holland, and Germany, compared with those which occur in Spain, Italy, the Mediterranean Islands—or still more so in Africa and the East and West Indies; and accordingly some authors (CRAIGIE) distinguish three varieties,—*e. g.*, (1.) The autumnal remittents of temperate countries, as England, France, Germany, Holland, Hungary. (2.) The summer and autumn remittents of warm countries, as Spain, Italy, Greece, the Mediterranean coasts and islands generally, the Levant, the north of Africa and Asia, and the United States. (3.) The endemic remittents of hot and tropical climates, as in the south of Asia, Central and Western Africa, Equinoctial America, and the West India Islands. Accordingly, remittent fever has received different names from the localities where it prevails. Thus we have the *gall-sickness* of the Netherlands, the Walcheren fever, fever of the Levant (IRVINE), Mediterranean fever (BURNETT), Hungarian sickness, *puka fever* of the East Indies, *jungle fever*, *hill fever* of the East Indies, *bilious remittent* of the West Indies and Mediterranean, Bulam fever, Sierra Leone fever, fever of Fernando Po and Bight of Benin, African fever, and Bengal fever. Prevailing on the borders of inland lakes, as in America, it is sometimes called the *lake fever*. (See page 31 on such nomenclature.)

Treatment.—With fever so various in its degrees of severity, it is not possible to do more than indicate the nature of the treatment which may be followed, as every special case must be

prescribed for and treated by its own special indications, and with a due regard to the nature of the prevailing epidemic.

The extent to which blood-letting can be carried, as recommended by Drs. Irving and Cartan and Mr. Goodison, will depend on the constitution of the patient, the type of the fever, the season, the climate, its immediate effect, and whether the prevailing epidemic is of such a kind as to be benefited by blood-letting. From the testimony of Dr. Hennen as to Corfu, Mr. Muir as to Cephalonia, Mr. Goodison as to Zante, and Mr. Boyle as to Sierra Leone, those who have long resided in these places do not bear blood-letting so well as strangers from colder and more temperate regions. When blood-letting is beneficial, its effect is in general to abate remarkably the pain, throbbing, and constriction of the head, and the pain of the orbits, to relieve epigastric oppression and tenderness, to render the pulse slower, less tense and oppressed, and to render the motion of the blood more free and less embarrassed. In some instances in which delirium is urgent, leeches applied to the occipital region are of the greatest benefit.

Local depletion over the epigastric region is often of great service, and enables the stomach to retain fluids and medicine. Purgatives are indicated to unload the alimentary canal, and to relieve the congestion of the visceral blood-vessels. The form most useful is the compound powder of jalap, with calomel, given in a bolus, and followed by three or four ounces of infusion of senna. Sometimes ordinary doses of purgatives have little effect till the local depletion has been effected over the region of the stomach; and it is also a good plan to change the purgative from time to time.

In every form and variety of the fever one of the most important guides in the treatment is to be derived from the nature of the prevailing disease, whether endemic or epidemic. Too much attention cannot be given to every means of knowing the type of the epidemic fever, whether *sthenic* or *asthenic*, and to study each individual case in relation to the prevailing type. First, the *duration of the stage of the fever* must be ascertained,—*i. e.*, whether it be of some hours' or of some days' duration, and whether, when the practitioner sees the patient for the first time, the actually existing paroxysm is at its accession or its decline. It is known by experience that the means of treatment

which would be salutary during the first few days cannot be used later to the same effect and in the same amount. There is less tolerance of remedies, and their effects are less therapeutic. Again, it is also known that the means which would arrest fever and save life, if applied at the accession of the paroxysm, would induce a dangerous collapse, or even destroy life, if applied at the stage of its decline, or towards its termination.

The various therapeutic agents which have been employed with various degrees of success in the treatment of remittent fevers are—*emetics, the warm bath, tepid and cold affusions, cold drinks, blood-letting, purgatives, diaphoretics, mercury, quinine or bark, arsenic, wine, and opium*. A review of the prominent modes of treatment of remittent fever, by the most eminent of British army surgeons, has led Sir Ranald Martin to make the following general remark:—namely, that a disease so varying in its nature, so general and complicated in its influence on the system, is not to be justly treated by one remedy. Bark and calomel, each a remedy of great power, will nevertheless not succeed in the cure of fever if used exclusively; and so it is with the most powerful of all means, blood-letting. Each remedy must therefore have its proper place in the treatment.

The first and most immediate object of treatment is to reduce the force and frequency of arterial action during the paroxysm. If the patient be seen in the forenoon of the first, second, or third paroxysm of an ordinary remittent fever, of *sthenic type*, and if he is of a sound constitution, and not beyond middle life, blood-letting from the arm, while the patient is in the recumbent posture, should be practised to the extent of relieving the sufferer from præcordial oppression, from visceral fulness and congestion, or from the intensity of the headache, whichever may predominate. The quantity of blood to be taken is to be regulated by the effects produced, and not by any arbitrary measure in ounces. Evidence of relief from visceral congestion is obtained from the following indications:—namely, reduced force and frequency of the pulse, reduction of morbid temperature, and gentle relaxation of the skin. This relaxation of the skin ought not to proceed to sweating, with further symptoms of depression of the vital powers. If it should do so from untoward circumstances, from half a grain to a grain of opium, or from fifteen to twenty drops of laudanum, with as many of chloric ether, should be administered, the object

of the administration of either of these medicines being to influence and soothe the heart's action, and to allay gastric or intestinal irritation; and it is only in cases of depression that opium is to be administered thus early in the treatment of the fever.

One general blood-letting will generally be found sufficient to relieve the patient from abdominal or cerebral oppression; and it will further have the effect of simplifying and rendering more efficient all the subsequent means of cure. Within an hour after the bleeding, a dose of *calomel*, with *compound extract of colocyath* and *James's powder*, should be given, followed in two hours by a powerful cathartic, such as *infusion of senna* with *sulphate of magnesia*. After the free action of these remedies, some degree of remission will be obtained in the afternoon, and the patient should be directed to take at bedtime from three to five or six grains of *calomel*, with four of *James's powder*, if the skin be dry; and during the past eight or ten hours he may have the free use of cooling drinks. On the early morning visit of the following day the remission will probably be more complete, when the *sulphate of quinia* alone, or in combination with the purging mixture, should be freely and repeatedly administered. Sir Ranald Martin recommends that it be given *with* the purgative mixture. By the forenoon the paroxysm may again recur in a milder degree, though to such an extent as to demand the application of leeches to the epigastric region, if any oppression or uneasiness exist there, or behind the ears if headache persist. A mixture composed of *antimonial wine*, with the *acetate* or *nitrate of potash*, should be given every two hours, so as to soften the skin and determine increased action of the kidneys. By these measures the daily decline of the disease is seen, and consequently there is a daily diminishing occasion for the use of active measures of cure, till, towards the fifth, sixth, or ninth day, convalescence is established.

If, however, remittent fever has existed unrestrained for several days, and the patient has not been seen till the accession of the third or fourth paroxysm, or even later, a general blood-letting is still the principal means of saving life, *provided the general powers of the constitution remain uninjured*; and it is to be followed by *calomel*, purgatives, and quinine, in the manner already indicated.

If the paroxysms have become indistinct, running into each

other, with brief or ill-defined intervals, while abdominal or cerebral complications arise, as indicated by epigastric fulness, or by approaching stupor or delirium, *blood-letting* may even now constitute the principal means to save life; but the blood *must be still more gradually abstracted* than before, whether generally or locally. Generally speaking, it is to be done by leeches, at the accession of the paroxysm. *Antimonials* are also to be used; cold must be applied to the shaved head; and while sinapisms and blisters must also be applied, on the influence of calomel chief reliance is to be placed, and the very first dawn of remission is to be seized upon to give quinine. We are not, in such cases, to wait for a clean tongue, the absence of heat of skin, or local complication. It must be given every *three* or *four* hours, with an occasional mild aperient in the intervals, until the dangerous symptoms shall have yielded—a result often observed to be coincident with the manifestations of the mercurial influence. Dangerous symptoms, such as those just noticed, will sometimes rise suddenly, without any loss of time on the part of the medical attendant, or neglect in treatment. If such symptoms are associated with yellowness of the skin, in persons broken in health, or of feeble constitution, or of dissipated habits of life, or who may have undergone much mental distress, the chances of a fatal termination are imminent.

When the spleen is enlarged, mercury is not to be used in the treatment of the fever; and blood-letting, either general or local, is not borne well. The blood is changed in such cases; it is more or less dissolved, and a general cachexia prevails.

The period of convalescence demands no less careful attention on the part of the medical attendant, especially as to diet and a timely removal from all malarious influences, by a voyage to sea or a change of climate. It is to the mismanagement of convalescence, and a too early discharge from hospital principally, that we must refer the numerous and fatal relapses in the fevers and dysenteries of our seamen and soldiers (MARTIN).

Regarding the method of treatment just described, my friend and colleague, Professor Maclean, writes in the following terms:—

“I have been led to take a view of the treatment of malarial fevers generally, and remittent fever in particular, differing from that laid down by many authors. It appears to me that the so-called

antiphlogistic treatment, so much insisted on by many writers, is based on the belief that the phenomena observed in a case of remittent fever are consequent on a process of inflammation. It is only on such a belief that antiphlogistic treatment can be justified.

“During the exacerbation of a remittent fever there is violent disturbance both of the vascular and nervous systems. Almost every organ, almost every function suffers,—the gastric intestinal membrane is affected, the liver and spleen suffer, the brain is involved, for rending headache and delirium are often present. Is it rational to suppose that an inflammatory process can be going on at one and the same time in all these various organs? Do the appearances observed *post mortem* give any support to such a doctrine? If not, on what principle can spoliative treatment be justified? Is it not rather the case that this terrible disturbance of so many organs is due to the presence in the blood of a subtle poison acting on them all? If so, surely the guiding principle of the physician in his treatment should be to counteract this poison, to neutralize it, or to expel it from the system, and so to prevent a recurrence of the exacerbation. This is the principle on which I have long acted, and I am satisfied that it is at once a safe and successful one. In quinine we have such an antidote—a therapeutic agent of unrivalled efficacy, which, if skilfully used, will rarely disappoint the expectations of the practitioner.

“It is always, of course, advisable to have the bowels thoroughly evacuated; and if the patient is seen when his stomach is loaded, it is well to evacuate its contents by an emetic. In ardent remittents, however, there is generally little call for this, as obstinate vomiting is almost always a troublesome symptom. This done, the period of remission must be watched for, and, the moment it arrives, quinine in a *full dose* should be given—not less than fifteen grains in the case of an adult. If the irritability of stomach be so urgent that the remedy is rejected, while measures must be adopted to allay it—such, for example, as alkaline remedies in combination with hydrocyanic acid, turpentine stupes, or even a blister to the epigastrium—time—precious time—should not be lost. Quinine should be given by the rectum in a full and efficient dose. By mouth or by rectum, or by both, quinine, in quantity sufficient to induce some of the symptoms of saturation (cinchonism), should be given before the time of expected exacerbation. According to my judgment and experience, it is bad practice to withhold quinine until an impression has been made on the force and frequency of the heart's action, from fear of increasing headache, causing congestion of organs, or the like. An impression on the force and frequency of the heart's action is best attained by arresting the paroxysm; and this is done most quickly, simply, and effectively by the early administration of quinine. I have

over and over again had patients brought to me from the malarial quarters of the city of Hyderabad, in whom it was impossible to distinguish any period of remission—the tongue black and dry, sordes on the teeth, the skin hot and parched, the pulse enormously quick, the intelligence feeble or gone—all pointing to a system so charged with malarial poison as to be well-nigh overwhelmed. In such cases quinine, with concentrated beef-tea and brandy, are urgently called for, and should be administered freely; and it is astonishing how men, by such measures, are often snatched from impending death. I have seen in a few hours consciousness return, a striking reduction in temperature, in the *frequency* of the pulse, with a remarkable accession of force and volume, follow the treatment indicated above. I do not advise, and never used quinine in the heroic doses advised by some. I have never exceeded a ʒ; but within such reasonable limits I have never seen it aggravate headache. On the contrary, I believe that in remittent fever—in fact, in all forms of malarial fever with which I am acquainted—I believe quinine to be a powerful remedy in quieting the tumultuous action of the circulation disturbed by the presence of this terrestrial poison. For some years past Warburgh's tincture has been much used in the treatment of malarial fevers in Southern India. It is a secret remedy, and therefore open to the objections very properly urged against all such remedies. It is understood that quinine enters largely into this remedy, and I do not doubt it. Be this as it may, I have given this 'tincture' a fair trial in some of the gravest forms of malarial fever, and it has also been extensively used by some of the most experienced officers of the Madras army; and I do not hesitate to say that I think it a valuable remedy. I have known it arrest at once some of the severest cases of remittent fever, no exacerbation appearing after the second dose. It almost invariably acts as a powerful diaphoretic—the most powerful with which I am acquainted. I have seen patients, under the influence of this remedy, saturate not only the bed-clothes but the very mattress, the patient's room and his person for days after giving out a strong odour of the medicine. For this reason it requires to be used with extreme caution, if at all, in the adynamic form of the disease. In urgent cases I follow the practice of the American physicians. I do not wait for a remission, but give quinine at once; and in all I am conservative of the patient's strength. I have seen violent delirium follow free leeching of the temples, and over and over again seen extreme and dangerous prostration follow depletive treatment, and that in cases where the violence of the disturbance indicated *power*; but these signs of power in the system are often most delusive, and, if combated by depressing measures, we must be prepared for sudden signs of collapse. Against the system of treating this fever by saturating the system with mercury I enter my

strenuous protest. I know nothing more deplorable than the condition of a patient whose constitution, already depressed by the presence of malaria, is further saturated by another poison which acts as a powerful ally of the first."

In the asthenic form of remittent fever, such as that so well described by Dr. Murchison as prevailing in Burmah, it is necessary to exercise great caution in depletion. All the cases he relates which had been freely bled exhibited the most aggravated typhoid symptoms, and most of them died. Even in the instance of young and robust recruits, low adynamic typhoid symptoms were sure to supervene in a short time after blood-letting; and, even although it gave temporary relief, it was certain to aggravate, if not to induce, the subsequent typhoid condition. If the headache is very severe and the pulse full, a few leeches may be applied to the temples at the commencement of the attack; but if the hair be cut short, or shaved off the scalp, cold lotions applied to the head, or the cold douche kept up for ten minutes at a time, gives great relief, and is the preferable remedy (MURCHISON). As soon as possible after the commencement of the paroxysm the bowels should be cleaned out with a purgative of calomel and compound jalap powder; or by colocynth, antimonial powder, and calomel. If typhoid symptoms betray themselves, stimulants, such as wine and brandy, must be given; but, as in intermittent fever, "quinine is undoubtedly the sheet-anchor," and it is best given, as in the former fever, in one large dose of twenty grains at the very commencement of a remission. Carbo-azotic or picric acid has been lately introduced as an active remedy in the treatment of malarious fever. Prepared by Calvert, of Manchester, it is of a light yellow colour; and in doses of two grains, cautiously repeated, it is to be pushed till the patient gets yellow-skinned.

MALARIOUS YELLOW FEVER—Syn., FEBRIS ICTERODES REMITTENS.

Definition.—*Febrile phenomena due to malaria, in which the exacerbation and remission are so connected that the fever resembles a continued fever, and is characterized by great intensity of headache and yellowness of the skin* (COPLAND,

DICKENSON, BOOTT); *but in which the urine is not suppressed, and continues free from blood or albumen.*

Pathology.—It immediately results from the history of yellow fever, that in its malarious form it is the product of the coasts of the West India Islands, the American equinoctial continents, several districts in Spain, and the west coast of Africa. All over the Caribbean Sea the disease takes place sporadically, or in insulated cases every season, more or less numerous according to the subjects and the number of new visitors, and there never is a season in which a few cases do not occur. At Vera Cruz, Havanna, and other towns on the Spanish Main, malarious yellow fever invariably attacks Europeans or Canadians who may land there between the months of May or June and October or November; but so long as such cases continue few, isolated, and sporadic, they attract no attention, and the disease is not heard of in ordinary years. It seems to prevail, for the most part, in towns situated on the sea or river coasts of alluvial countries in warm climates; and that, while the banks of these rivers or seas are liable to occasional alternate periods of inundation and drying up, the fluctuations of the tides, co-operating with these, contribute powerfully, under intense solar heat and a windless atmosphere, to render the towns along the shores of such districts the seat of malarious yellow fever. “While ague is the offspring of the marsh or its margins, and remittent is the effect of a more concentrated form of the same exhalation from some moist surface in the process of solar desiccation, the malarious form of yellow fever appears to be the product of that state of the atmosphere which takes place after a long continuance of solar heat, with little or no wind, in those points chiefly where the atmosphere of the sea and that of the land are in constant communication and interchange. It is, indeed, a remarkable fact that the intense form of remittent fever which has been distinguished as malarious yellow fever, and sometimes as ‘bilious remittent of malignant type,’ is rather rare in the interior of countries, and is seldom found in towns, situated on rivers, higher than the influx of the tide. The fevers which appear in these situations are more of the usual remittent character; and in the interior of the American continent there is little doubt that the *lake fever* represents the malarious *yellow fever* of the coasts. Even in Europe, while the towns on the sea-coast and on rivers were labouring

under the malarious yellow fever, the sickliness in the interior approached more to that of the remittent or remittent continuous type" (CRAIGIE). For this reason the term *litoral*, as well as *paludal*, is used to designate this class of fevers.

The endemic conditions under which the malaria give rise to this form of yellow fever might be referred,—(1.) To thermometric temperature of the air; (2.) To the state of the atmosphere as to currents of winds and electricity; (3.) Local peculiarities of surface already referred to; (4.) Constitutional susceptibility, and crowding together of masses of people.

That intense solar heat contributes greatly to the development of the yellow form of malarious fever, is shown by the situations of those parts where it is peculiarly endemic, in relation to the prevailing temperature. Thus, it is found to prevail chiefly in places situated in the eastern regions between 10° of south latitude and 42° of north latitude. On the continent of Europe it has generally prevailed in places situated between the 36° and 38° north latitude, and has never gone farther north than Barcelona on land, or in latitude 48° north on the sea. That it has gone farther north, it has been alleged, but the authenticity of the statement is doubted. In these northern latitudes it is also observed that the malaria of yellow fever cannot pass over a thousand yards of water without being deprived of its power.

The following observation was made by Sir John Pringle on the fevers of Walcheren and South Beveland in 1747:—

"These epidemic fevers, by reason of the great heats of the season, not only began more early than usual, but were fully as fatal to the natives as to us. But Commodore Mitchell's squadron, which lay all this time at anchor in the channel between South Beveland and Walcheren, in both of which places the distempers raged, was neither afflicted with fever nor flux, but amid all that sickness enjoyed perfect health—a proof," he says, "that the moist and putrid air of the marshes was dissipated or corrected before it could reach them" (*Diseases of the Army*, p. 58).

The very same observation was made at the very same spot, fifty-two years afterward, by Sir Gilbert Blane:—

"I had, in the course of this service (at Walcheren, in 1809), an opportunity of observing the extent to which the noxious exhalations extended, which was found to be less than I believe is generally known.

Not only the crews of the ships in the Road of Flushing were entirely free from this endemic, but also the guard ships stationed in the narrow channel between this island and South Beveland. The width of this channel is about six thousand feet; and although some of the ships lay much nearer to the one shore than the other, there was no instance of any of their officers or crew being taken ill with the same disorder as that with which the troops on shore were affected" (*Med.-Chir. Trans.*, vol. iii., p. 27).

It is now also generally believed that the malarious form of yellow fever cannot exist except in places where the average range of temperature is high throughout a considerable part of the year; and for this reason it is believed that it will not become a disease of this country. Sir Gilbert Blane asserted that it never appeared either in tropical climates or in the temperate latitudes, unless when the atmospheric heat has been for some time steadily at or above 80° Fahr., 21° of Reaumur, or 26.67° Cent.; according to Humboldt, 75° of Fahr., or 24° Cent.; and according to Matthei, 72° Fahr., or more. The disease is also found not to prevail in mountainous situations. According to Humboldt, it has never ascended to 3,044 feet above the level of the sea, and according to Sir Ranald Martin, never above 2,500 feet; and below the former limit the Mexican oaks do not flourish, showing that the constant average temperature below this is of a tropical character. In Jamaica, according to Dr. Craigie, it rarely ascends 1,600 feet above the level of the sea (Dr. Lawson's instance of the outbreak at Newcastle being considered an instance of "specific yellow fever"). "In Jamaica the medium temperature of Spanish Town in the hottest months is about 85° Fahr., or between 83° and 85° ; and in Kingston it is much the same, ranging from 85° to 90° , and rarely falling below 80° from May to the end of September. At the more elevated parts, however, the temperature diminishes, being only about 70° at Stoney Hill, elevated about 1,300 feet; at Cold Spring, 4,200 feet above the level of the sea, only 60° ; and at the summit of the Blue Mountains, which are estimated to be, 7,200 feet above the level of the sea, the thermometer is found to range in August from 47° at sunrise to 58° (HUNTER) at noon: or at an average of 60° (MOSELEY).

At Stoney Hill, the first of these places, yellow fever has sometimes, though not very often, displayed its epidemic virulence in a very bad form.

In the Island of Trinidad, however, the ridge behind Port of Spain, which is a limestone rock elevated 1,500 feet above the level of the sea, has been highly productive of yellow fever, and has cost the lives of many men in attempting its clearing and fortification.

The composition of the soil has been believed to exercise some considerable influence on the production of malarious yellow fever. On this subject, however, the facts are discordant. Alluvial soils are those where malarious yellow fevers have mostly prevailed, as at Grenada, St. Domingo, New Orleans, Philadelphia, New York, Boston; or calcareous, as in Jamaica. It has also been observed that a beach, bank, quay, or wharf is the place where the disease first makes its appearance, when such beach, bank, quay, or wharf is alternately immersed in sea water and exposed to the drying effects of great solar heat. The drying effects of great solar heat have also been supposed to extricate some deleterious material from the green wood of new ships (WILSON), and also from forests of mangroves (INGRAM HUMBOLDT, WILSON).

In all those localities where the disease is endemic it seems to manifest a decided preference for the natives of the colder regions. Thus the British, Germans, Swedes, Danes, are more liable to suffer than Italians, French, or Spaniards; and in ordinary years the natives, and especially the coloured population, are rarely attacked.

The diagnosis between the specific contagious yellow fever and the malarious form of yellow fever is at all times difficult; and severe marsh fevers in certain geographical limits have a close resemblance to contagious yellow fever. But they are not contagious, and urinary and blood symptoms do not occur in them. It must also be remembered, in accounting for its origin in any case, that marsh fevers have become developed weeks, and even months, after exposure to the exciting cause.

With regard to the further history of the phenomena and treatment of this form of fever, the reader is referred again to what is said under remittent fever, and simply stating that when an observer has seen only the milder form of marsh remittent fever, and is then suddenly called upon to witness an attack of malarious yellow fever, he may well believe that the affections are entirely distinct. But after a time, when the intermediate forms have been more closely scrutinized, it is found that at no



